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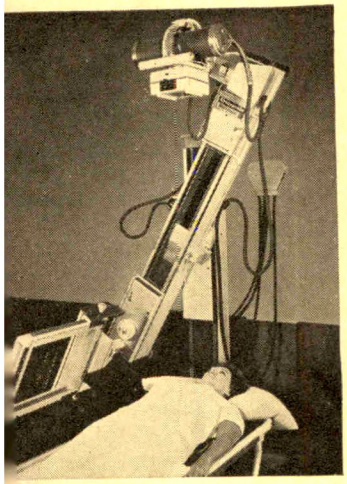
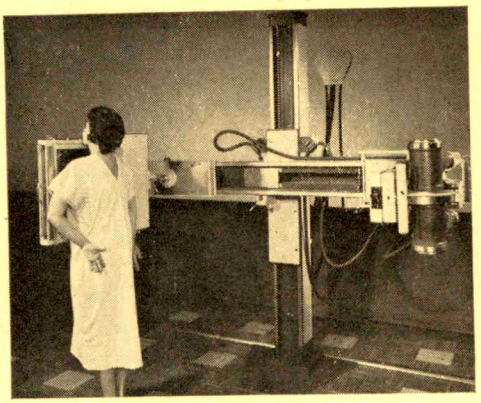
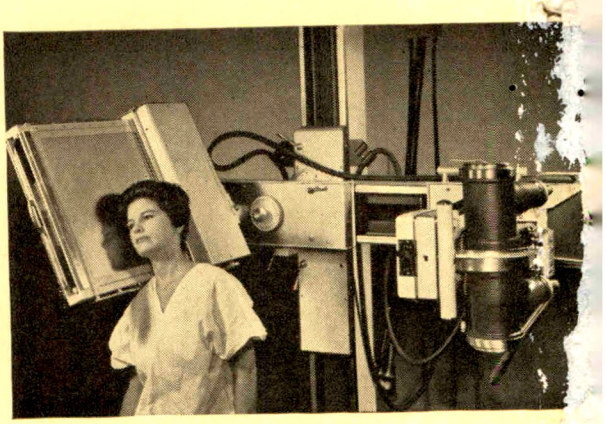
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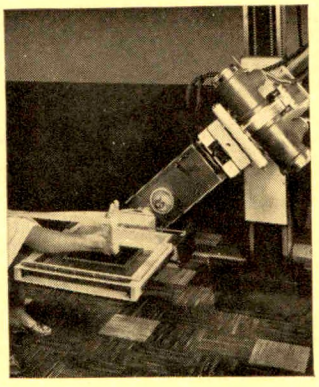
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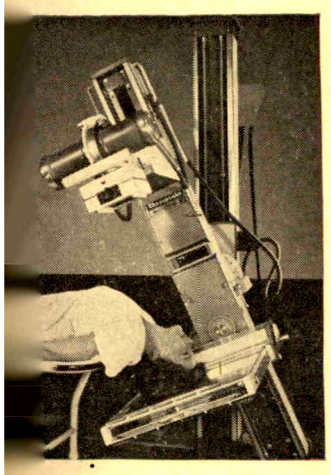
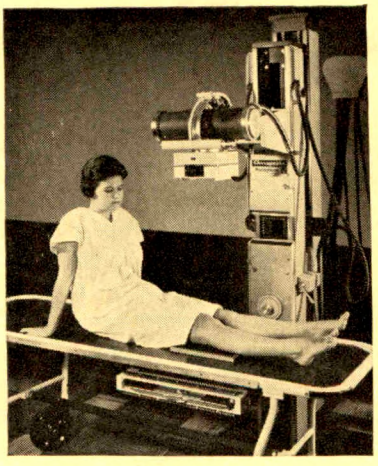
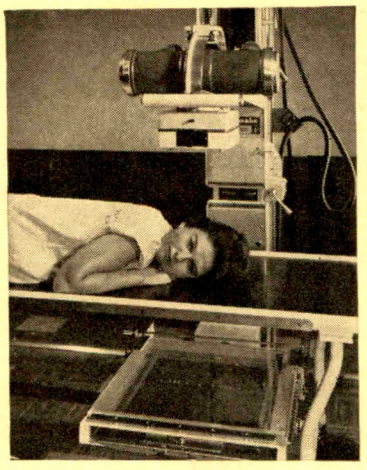
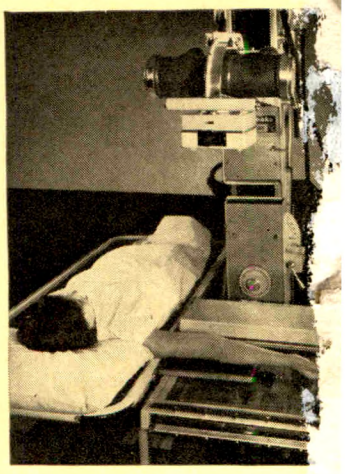
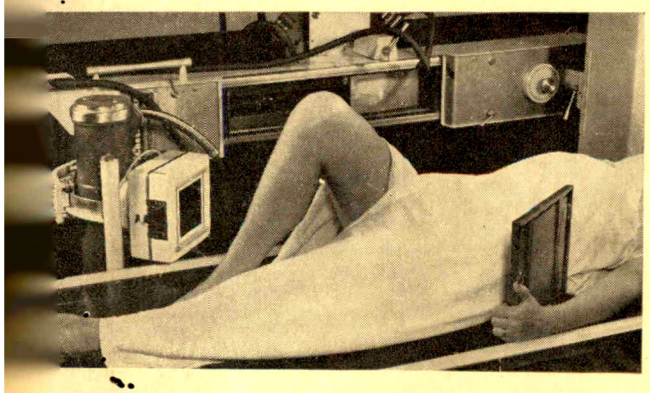
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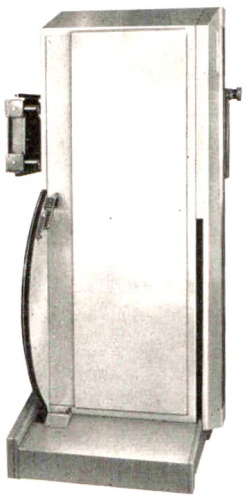
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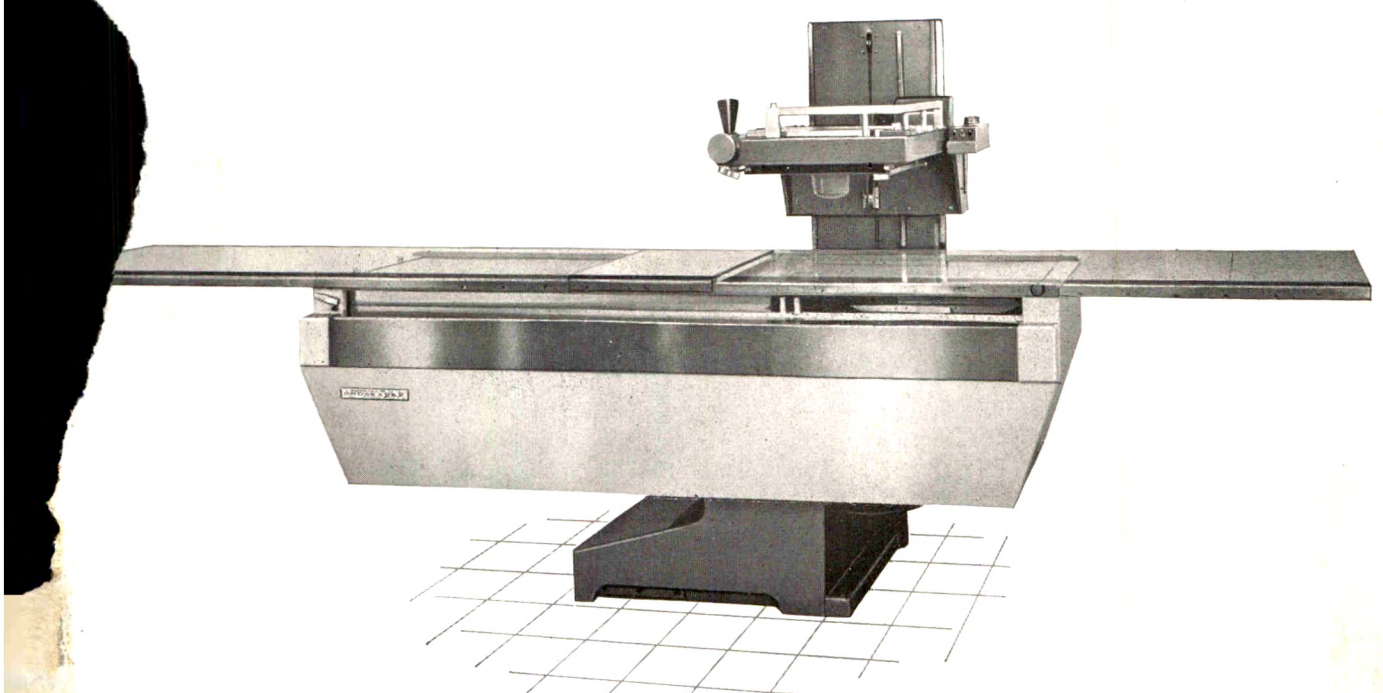
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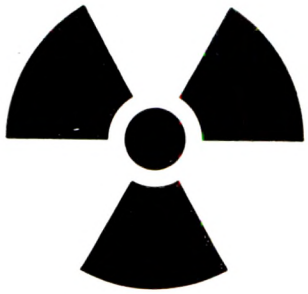
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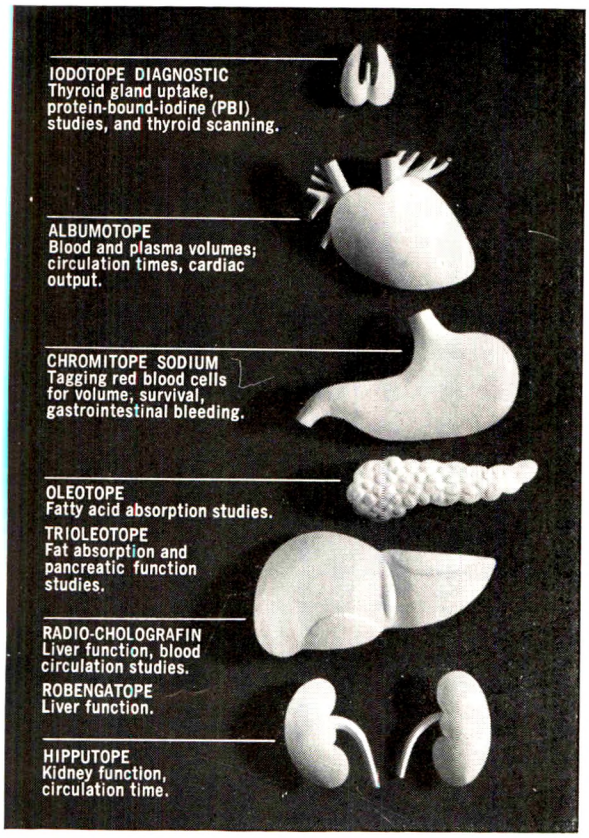
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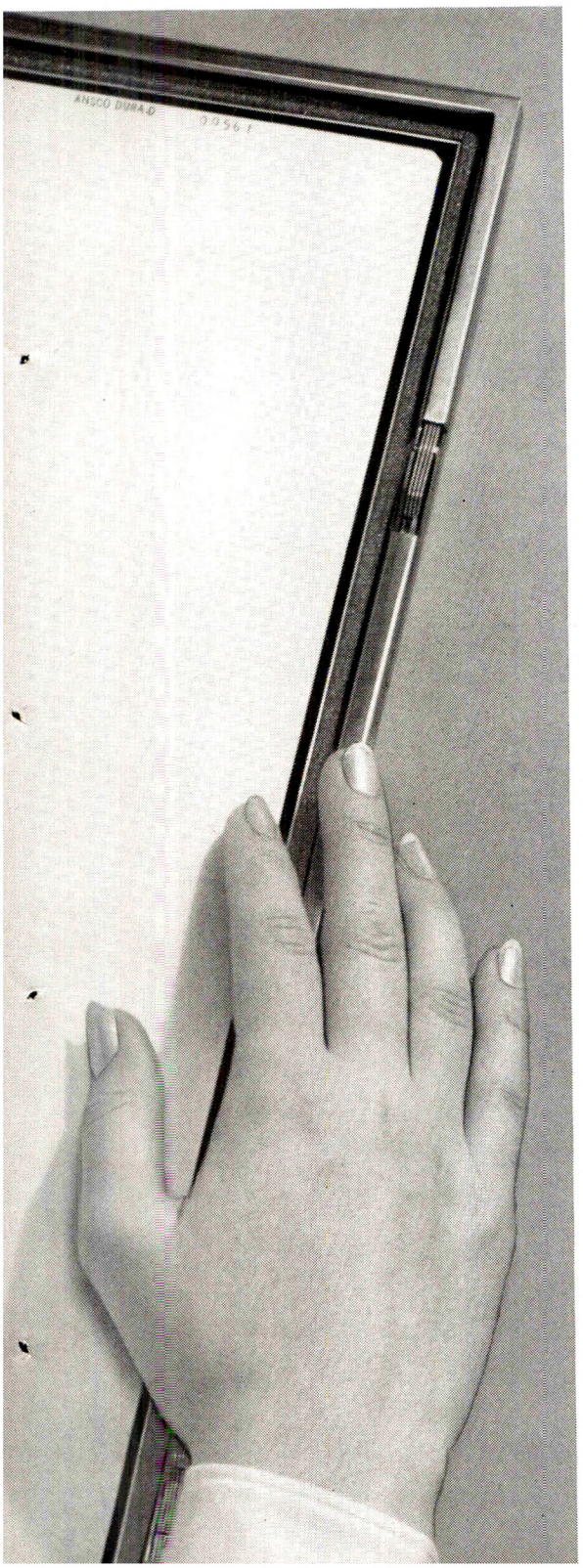
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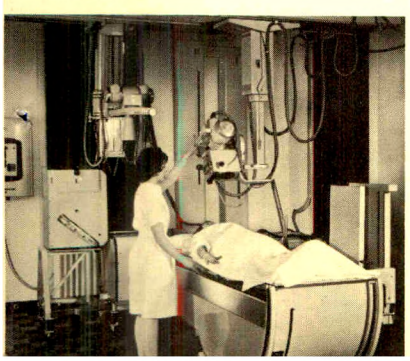
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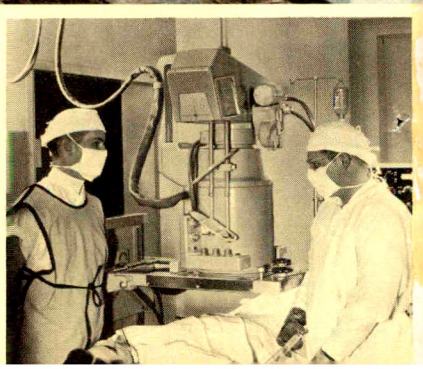
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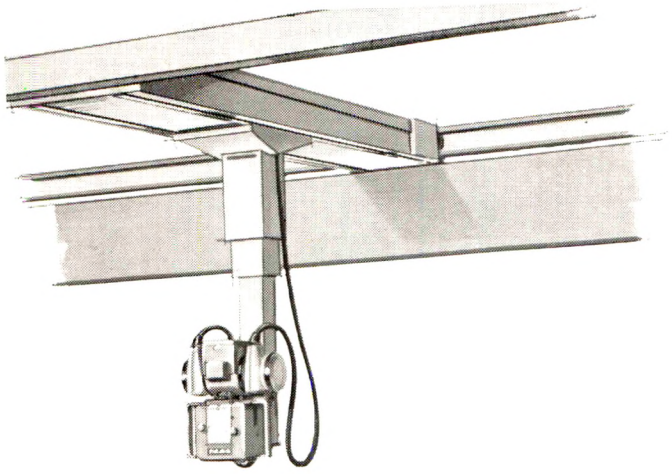


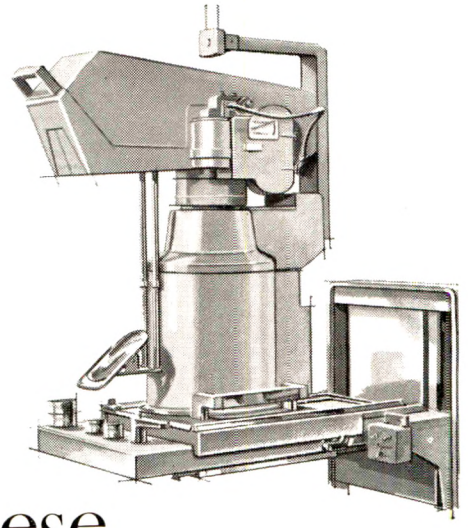
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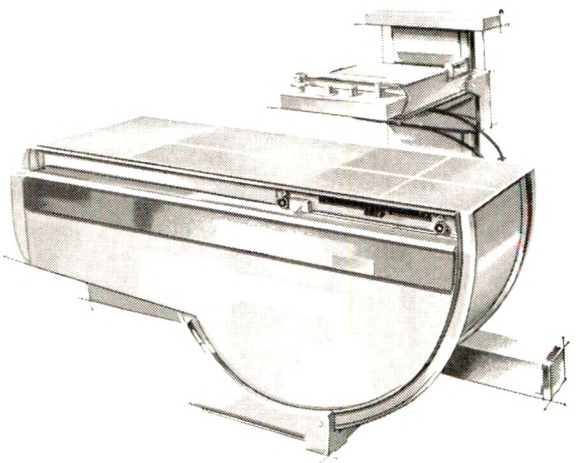


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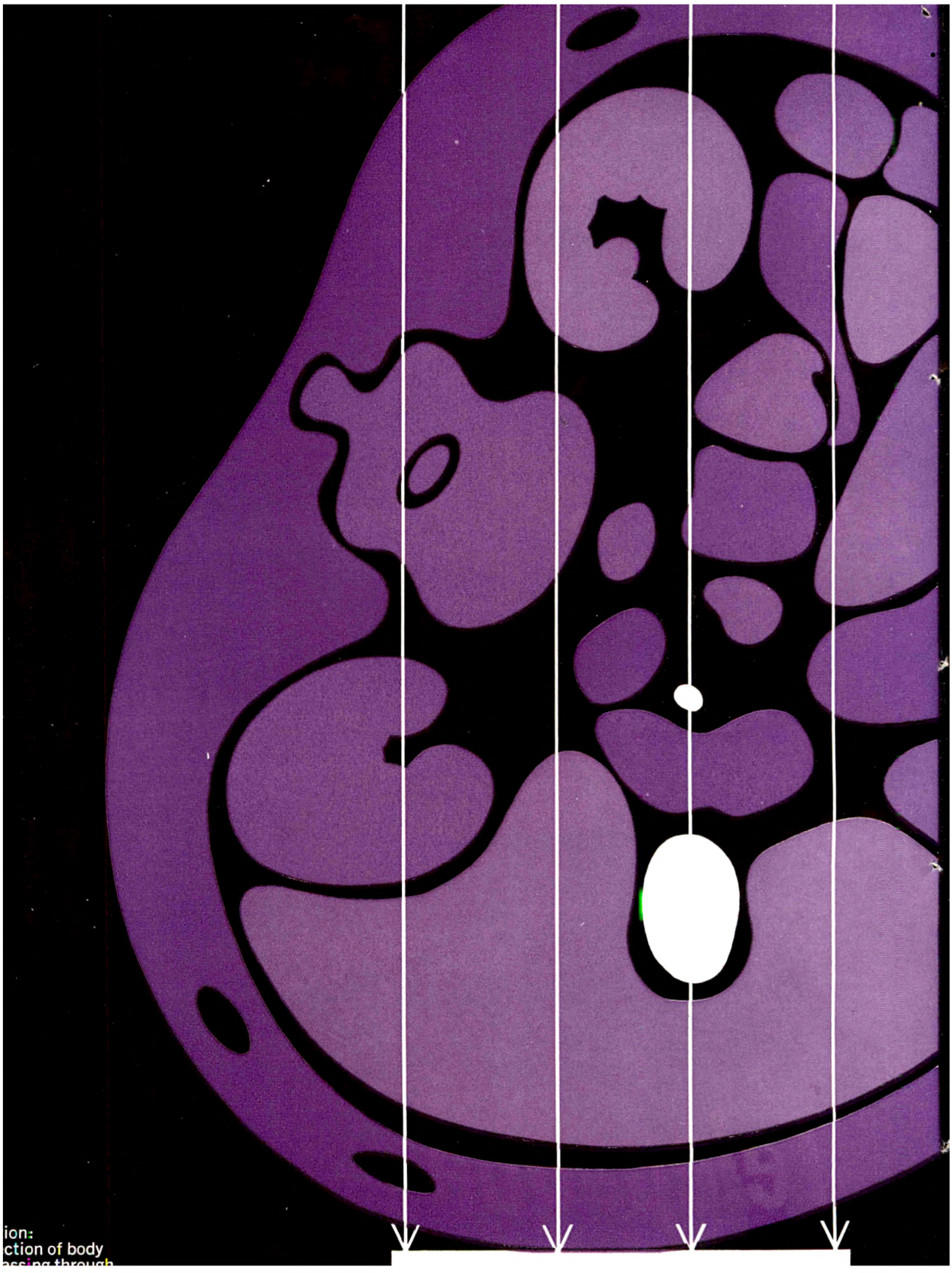
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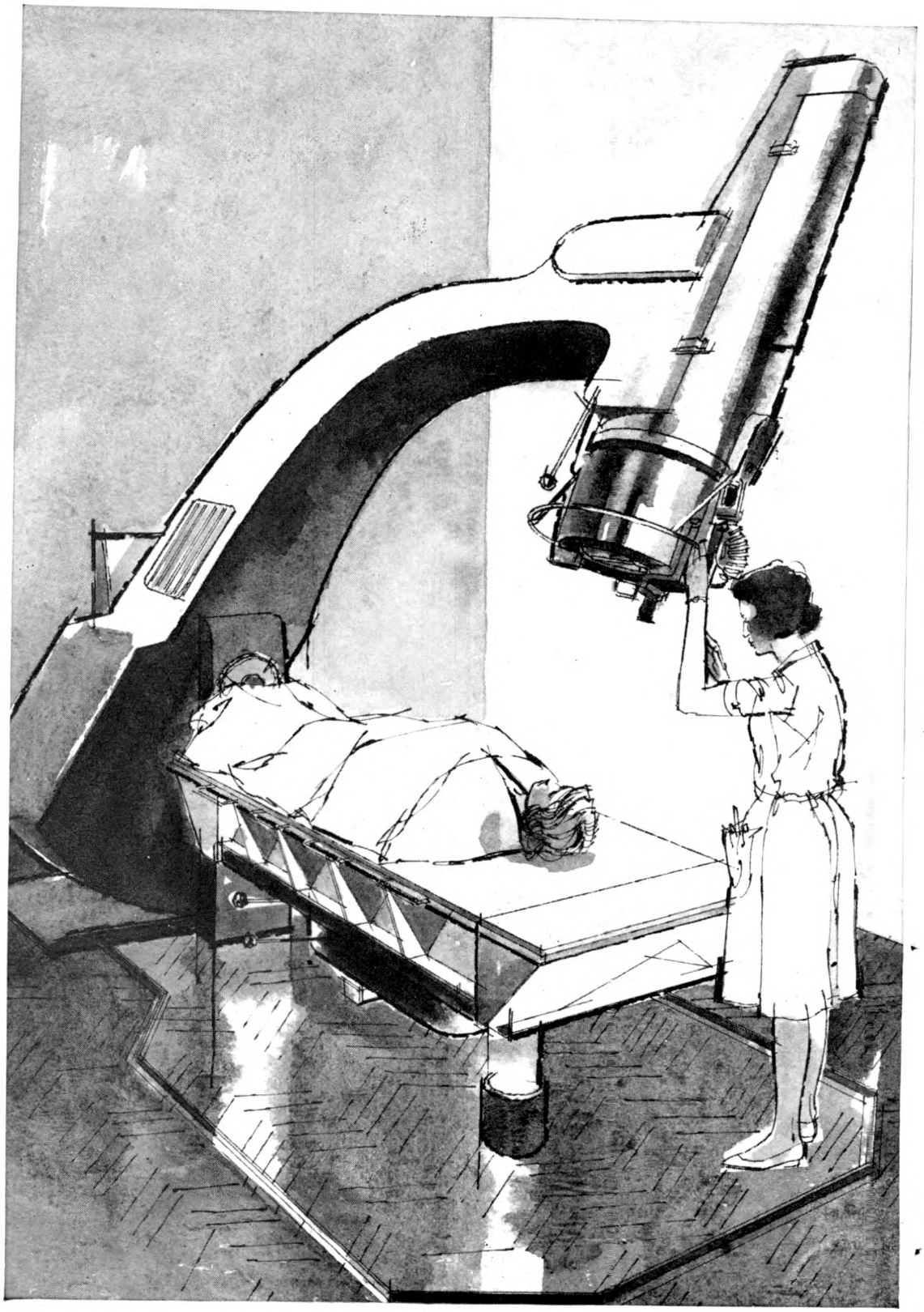
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1. Whitehouse, W. M.: Iopanoic acid, Ann. New York Acad. Sc. 78:809, July 2, 1959. 2. Baker, H. L., Jr., and Hodgson, J. R.: Further studies on the accuracy of oral cholecystog-



in service

THE VICKERS SUPERVOLTAGE X-RAY THERAPY UNIT



This is the heart of the Unit: the sealed-off accelerator tube.

Vickers 6 MeV X-band Linear Accelerators are now in regular service at the General Hospital, Newcastle-upon-Tyne and Guy's Hospital, London. Other units have been supplied to the Westminster and Royal Marsden Hospitals, to be followed by installations at St. Bartholomew's Hospital, London, The National Institute of Radiological Science, Japan, and Queen Elizabeth's Hospital, Kowloon.

Advanced technical design has achieved the following features.

* High Dose Rate with simplicity of control.
* Rapid set up by a single operator with minimum physical effort — all movement of the servo-assisted gantry and motorised field diaphragms are controlled from the X-ray head.
* High positioning accuracy with the aid of optical front and back pointers.
* Unique sealed-off accelerator tube for reliability of service.
* Only tested high quality components are used.
* Cleanness of line and overall compactness obviate the need for specially constructed buildings.

Miniaturisation of the modulator and the use of the X-band frequency has enabled all the equipment to be housed in the gantry and control unit, without the need for a separate apparatus room.

Further information can be obtained from:

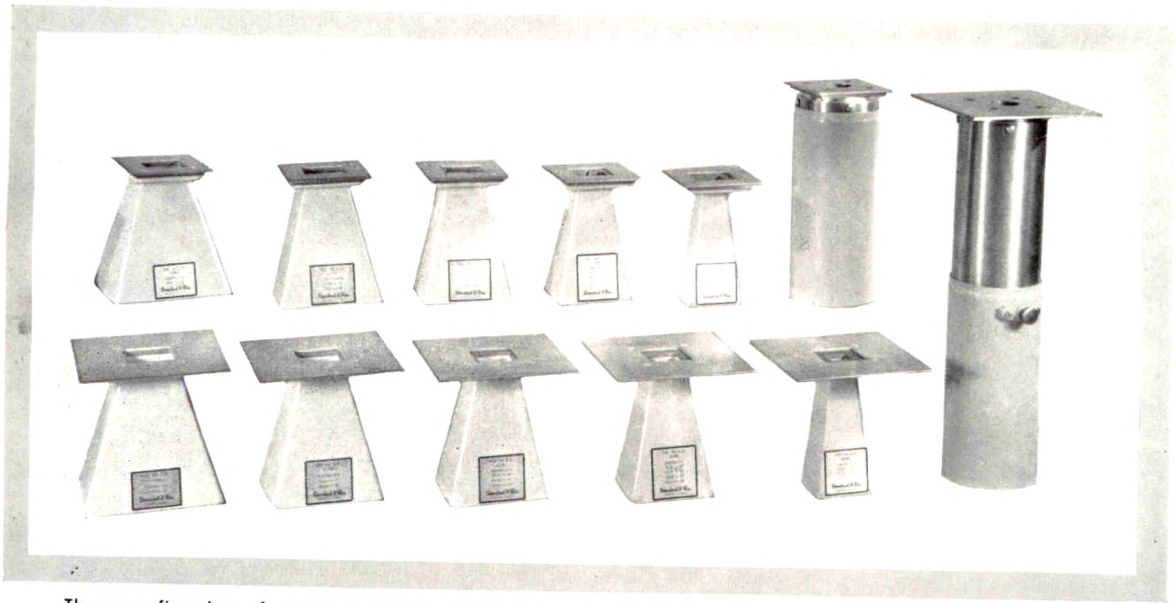
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Meet Our Brand New Family of Modern Standex DOUBLE DIAPHRAGM CONES

- **the one and only answer to FULL PROTECTION for the patient***
- **its power to limit radiation is unmatched, unequalled by any other method***

*In a recent report developed jointly by NEMA and the Radiological Society of North America, it is stated that not only is there a "great deal to be said for the use of fixed type cones" but *"also this type of cone permits the reduction of stray radiation to lower values than are obtainable with the use of adjustable diaphragm cones."*



There are five sizes of *rectangular double diaphragm* cones—a size to cover all standard size films at conventional focal-film distances. There is also a 4" diameter, 10" long round cone and a 4" dia. 10" to 18" adjustable extension cone. All cones are available with either a 4" x 4" or 6¼" x 6¼" base plate. Other types and sizes of bases can be adapted to all these new cones.

Even with the present popularity of radiographic collimators, fixed radiographic cones are still preferred by many radiologists and technicians. There is and will be for a long time to come, a continuing demand for fixed radiographic cones that offer present day protection requirements.

We have re-designed our line of radiographic cones and upgraded built-in protection to meet the requirements of the Bureau of Standards (Handbook #76) for *150PKV* operation. Our new line of cones has built-in protection of at least 4¾mm *lead* (3/16") NOT lead equivalent—full protection against the primary beam.

Your Standard representative will be glad to supply any additional information desired, quote prices and delivery of Standex Cones to meet your needs.

STANDEX DOUBLE DIAPHRAGM RECTANGULAR RADIOGRAPHIC CONES

CONE NUMBER	NO. 30	NO. 36	NO. 40	NO. 48	NO. 72
FILM COVERAGE (Approx.) AT 30 INCHES FFD.	14x17	11x14	10x12	8x10	5x7
FILM COVERAGE (Approx.) AT 36 INCHES FFD.		14x17	11x14	10x12	6.5x8.5
FILM COVERAGE (Approx.) AT 40 INCHES FFD.			14x17	11x14	8x10
FILM COVERAGE (Approx.) AT 48 INCHES FFD.				14x17	10x12
FILM COVERAGE (Approx.) AT 60 INCHES FFD.					11x14
FILM COVERAGE (Approx.) AT 72 INCHES FFD.					14x17

The chart above shows how you can specify the size Standex Rectangular Double Diaphragm Cones to meet your requirements. All Standex Cones have specifications attached, clearly showing the field (film size) covered at various focal-film distances.

Standard X-RAY COMPANY
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high speed with high definition

in a new and superior intensifying screen!

ILFORD FAST TUNGSTATE SCREENS

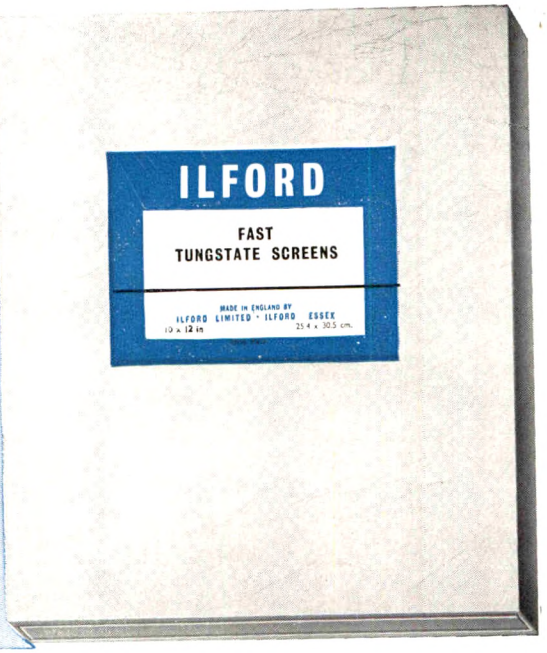
These new screens achieve a combination of qualities never before available. They are the result of over 35 years of Ilford experience in manufacturing high quality intensifying screens. Specifically, Ilford Fast Tungstate Screens offer:

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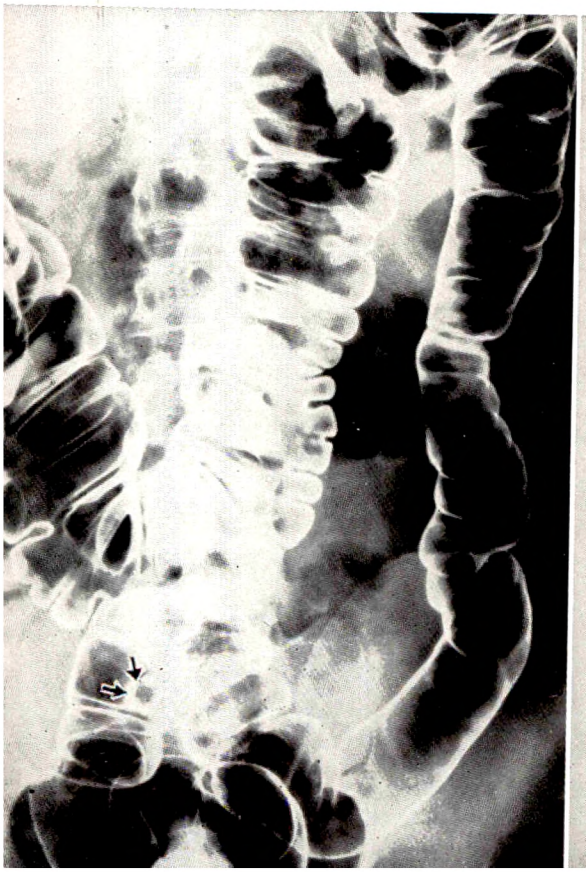
...radiological studies with Barotrast
are routinely exceptional...

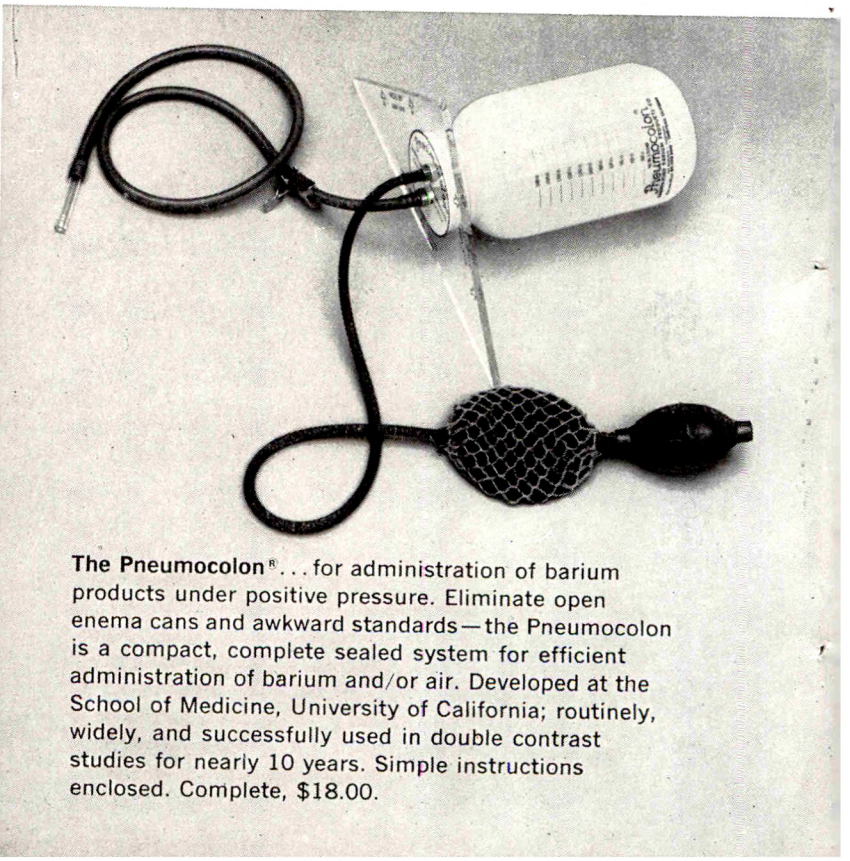
*BAROTRAST MAINTAINS UNIFORM SUSPENSION

Barotrast will not separate, crack, peel, or clump.
Its unique adherence to the intestinal mucosa gives a uniform, stable, and flexible coating.
It possesses unparalleled properties of flow, coating, and water retention which are not affected by mucin content, enzymes, or changes in pH.
Recognizing these singular benefits of Barotrast, many radiologists who have already adopted its use for double contrast studies are now finding Barotrast to be exceptional in upper GI and small bowel studies.
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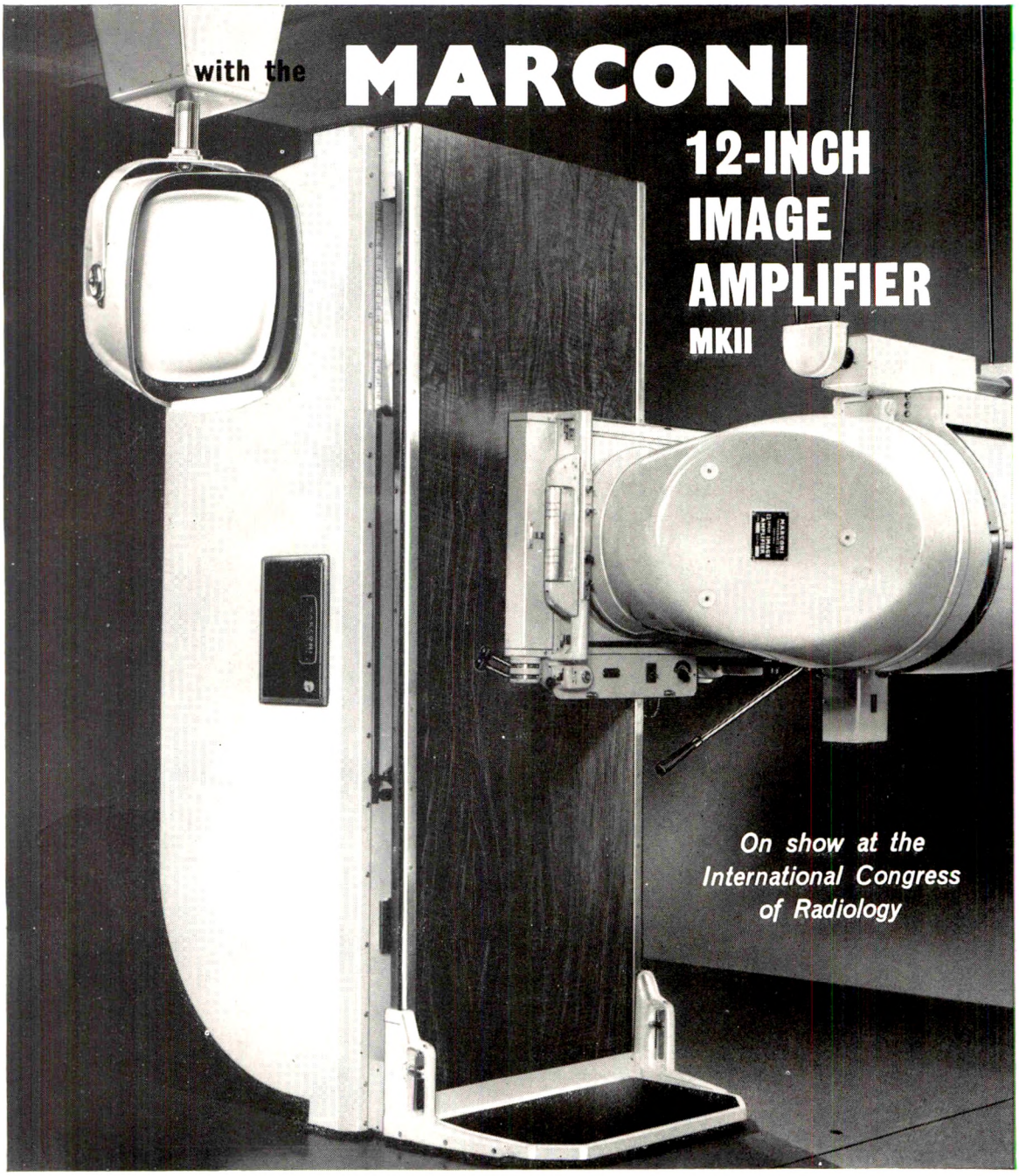
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Barium held in suspension continuously by a special mixing unit.

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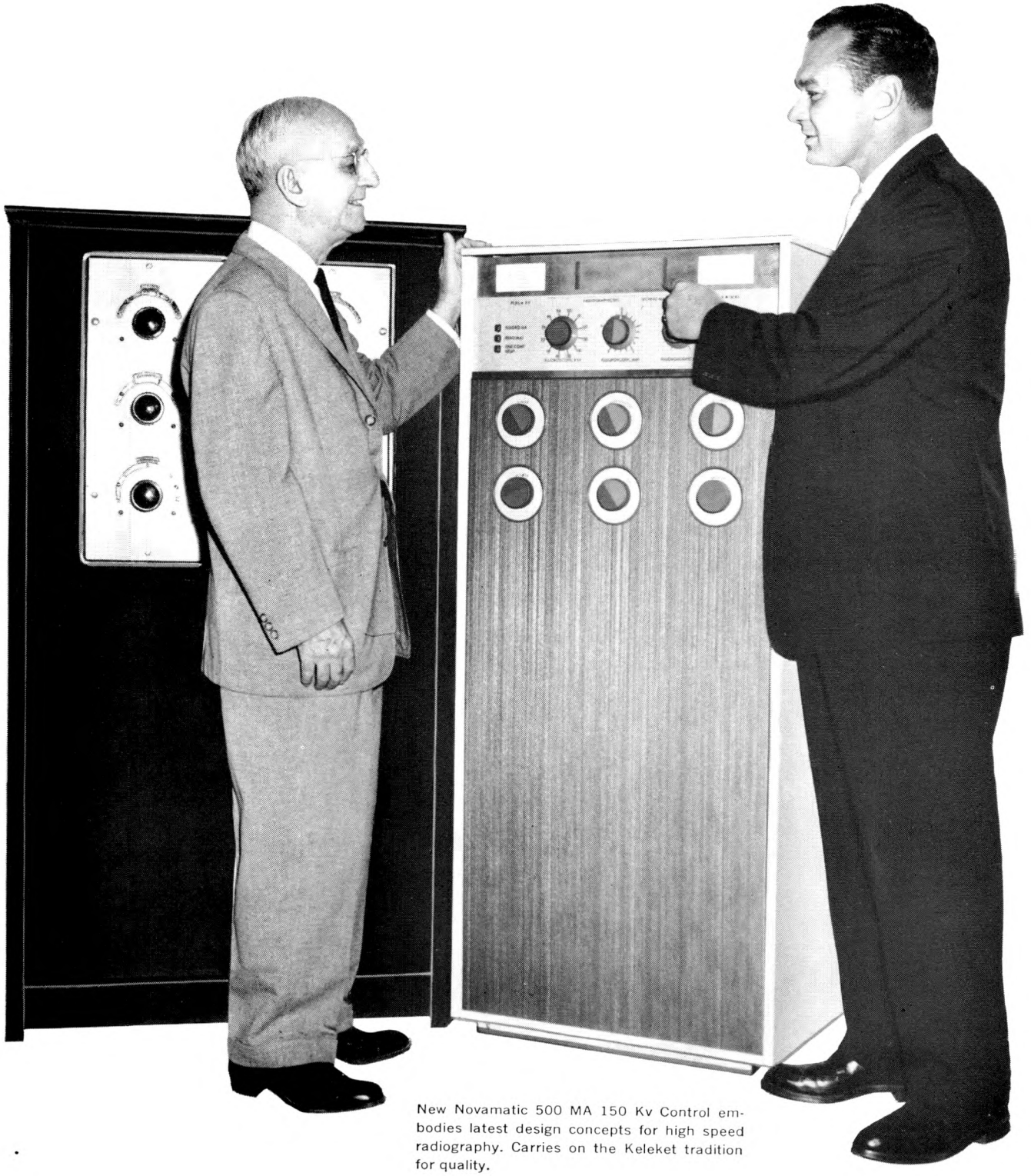
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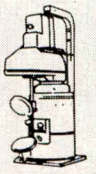
New Novamatic 500 MA 150 Kv Control embodies latest design concepts for high speed radiography. Carries on the Keleket tradition for quality.

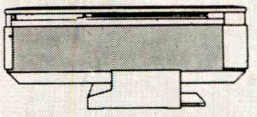
a tradition of dedication

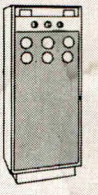
The lure of the x-ray business runs strong. Forty-eight years ago R. P. Kincheloe, Sr. entered the field and for five years was chief engineer of Keleket. In 1919 he was appointed the Keleket X-Ray distributor for the entire southwest United States. His company now serves leading radiologists and institutions in Louisiana, Texas, Oklahoma, and Colorado. So strong is the Kincheloe family feeling for service, that Dick Jr. entered the business, and is now Vice President of R. P. Kincheloe Company. The technical qualifications of the Kincheloe organization are outstanding, beginning at the top: R. P. Kincheloe, Sr. is a graduate electrical engineer from the University of Cincinnati, and Dick Jr. is a graduate electrical engineer from Rice University. The Kincheloe's ability and dedication is typical of the Keleket spirit.

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OF PRESENT DAY BRONCHOGRAPHY*

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DIONOSIL

widely used, emphatically preferred

Fifty-seven specialists in thoracic medicine and surgery, mostly in the United States but some practicing in Europe, Canada, South America and Japan, replied to a questionnaire sent out by the Committee on Bronchoesophagoscopy of the American College of Chest Physicians.

The results have been analyzed and published.

Of these 57 specialists, no fewer than 47 used Dionosil.

Many used Dionosil exclusively, and emphatically preferred it. Particularly praised was Dionosil Oily, described as "certainly the favorite preparation at the present time".

The survey showed that:

- Bronchography is increasingly used to elucidate lung conditions other than bronchiectasis.
- Use of Dionosil instead of iodized oil eliminates risk of iodine sensitivity.
- With Dionosil alveolar filling is quite unusual.
- Complications are rare with Dionosil.

**Diseases of the Chest,* (1958), *33,* 251

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is available as oily or aqueous suspensions of propyliodone, in 20 cc vials and in 100 cc bottles

Dionosil is a product of GLAXO Laboratories, Ltd., Greenford, England, distributed in U.S.A. by

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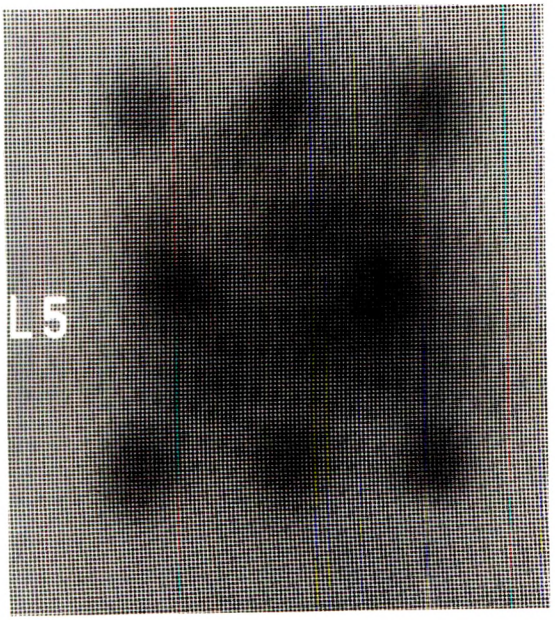
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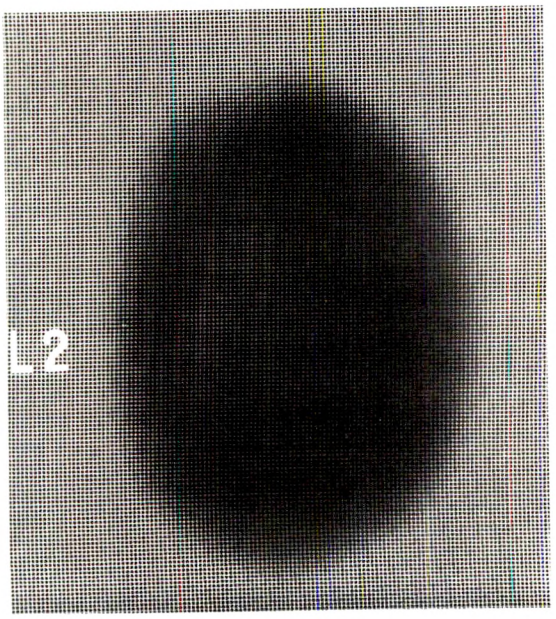
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X-RAY NEWS

No. 57

DIFFERENT WAYS OF CHECKING FOR POOR SCREEN CONTACT





by **Raymond W. Horner, R.T.**
Chief X-ray Technician
School of X-ray Technology
Lower Bucks County Hospital, Bristol, Pa.

As in every large and busy X-ray Department, a certain number of our films are found unacceptable because of blurring. This blurring was often interpreted as coming from motion and the patient was re-examined. Usually, a different cassette was used and the repeat examination was sharp and of sufficient quality for interpretation.

After a period of time it became apparent that some of the blurred areas followed a certain geometric pattern and further, that the blurrings were peculiar to certain cassettes. After this was noted, and after all other possibilities were ruled out, the question of poor screen contact was considered. We then found that most of the blurrings previously ascribed to motion were indeed coming from poor screen contact. The similarity in appearance between the two is marked and, therefore, should be strongly considered in all cases of unexplained blurring.

Following are four different methods of testing screen-film contact. The first method is to place a piece of wire screen on the face of the cassette and make a test exposure. Second method: The wire screen is enclosed between two pieces of cardboard

---

Photo L5. Here are a number of smaller areas of poor contact—each caused in the same way as the single large area in Photo L2 and each is subject to the same cure. Small areas of poor contact are especially easy to miss, and it is here, particularly, that close inspection, tilting and distance help.

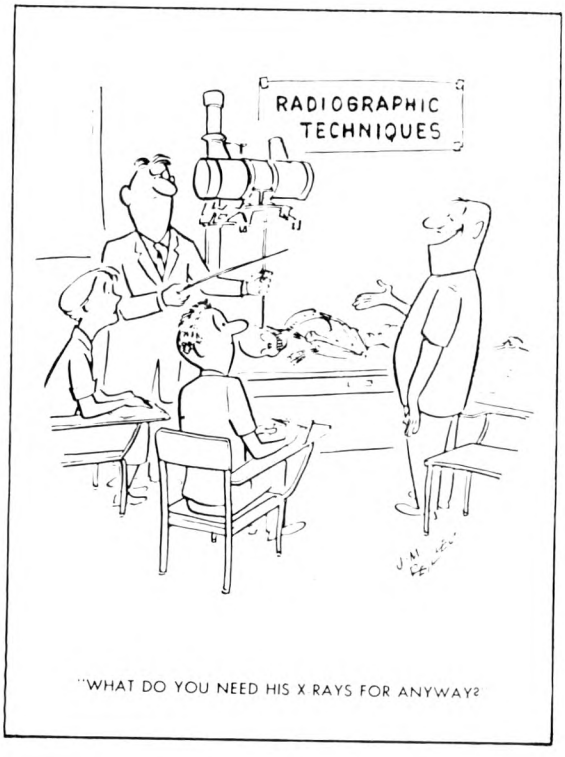
Photo L2. This demonstrates a large area of poor screen contact, which is clearly seen as a darker oval extending across the middle of the radiograph. Use the radiograph to cut a thin piece of carboard to the shape of the area, then fasten this behind the screen to correct the poor screen-film contact.

OVER

and the test exposure is made. Third method: Place the wire screen between the bakelite front of an old cassette and a piece of cardboard backing ¼ inch thick. Then make exposure. The fourth and more expensive procedure is to place the screen between two pieces of bakelite (the cost being approximately seven dollars for the screen and bakelite).

After tests of various cassettes were made, it was found that the areas of poor contact were much more visible when the test films were viewed with the red glasses used for fluoroscopic accommodation. In addition to accentuating the areas of poor contact, this method is much easier on the eyes. It was also found that the areas of poor contact were more visible when test films were viewed from a few feet back. Viewing test films from an angle, rather than head-on, also helps make the areas of poor contact more prominent.

Because of the simplicity and dollar saving in preventing re-examinations, I feel that it is essential for every department to have a method of testing screen contact. If the same blurred area turns up in the same cassette in a series of studies, poor contact should be suspected and the cassette tested for screen contact. In addition, a great deal of unnecessary patient exposure is avoided by making sure that your screen contact is good. Testing equipment is inexpensive and the testing procedure is simple. We, as technicians, should be aware of the problem of poor contact, and know how to correct it when we see it.



"WHAT DO YOU NEED HIS X RAYS FOR ANYWAY?

Tips for Technicians

The use of Hypaque* Sodium in small bowel studies

by **Beatrice Toensing (RT)**
University of Minnesota Hospital
St. Paul, Minnesota

We have been adding Hypaque Sodium powder to our solutions of Barotrast* * or Veri-o-pake* * *for small bowel series. This addition, we have found, cuts the follow-up time from the average four hours to about 45 minutes.

In using the powder for small bowel studies we add two tablespoons to a 200 cc glass of barium mixture—either the regular mixture of 1 part barium to 2 parts of water or, with Veri-o-pake, 2 parts barium to 1 part water. This makes a solution about the consistency of a milk shake.

Our films are taken with the high KV technique at 20 MAS (12:1 GRID). Films are taken sooner than with the old method. First the abdomen film taken immediately after the routine GI series, then a second taken 10 minutes following, a third twenty minutes later and a fourth in half an hour.

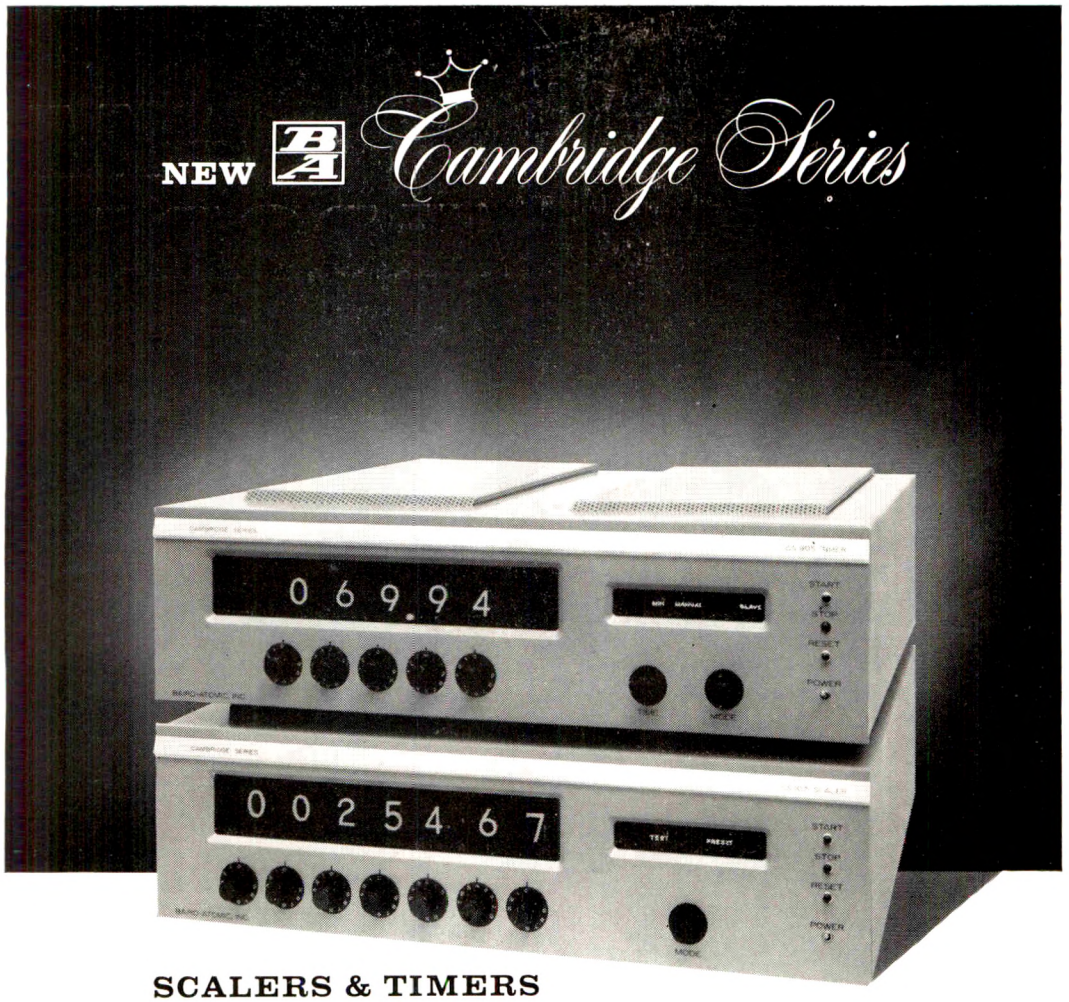
An alert radiologist can pick up the slight difference in density, if he studies several series with and without Hypaque, for comparison. It will be noticed that on the first two films the Hypaque moves faster than the barium.

At present, we are also adding Hypaque powder to the formula for infants on our routine GI series to make a 40% solution, in place of barium sulfate. This is particularly helpful in avoiding the constipation that usually follows when barium is used for children.

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The NEXT STEP in Teletherapy

—AECL's ELDORADO 6



The revolutionary NEW THERATRON 60 and ELDORADO 6 will be on display at the International Congress of Radiology, Montreal, Canada, August, 1962.

May we have the pleasure of demonstrating the many new and desirable features of these superb yet low cost advances to the field of Cobalt 60 Teletherapy.

The most remarkable advance in Cobalt 60 Teletherapy since AECL pioneered the first units more than a decade ago.

The THERATRON 60 and the stationary unit, ELDORADO 6, have been designed to meet the needs and ideals expressed by leading Radiotherapists and physicists from all parts of the world in consultation with AECL.

One of the most remarkable results achieved is the great reduction in cost in spite of advanced design and styling and the many significant improvements that encompass every aspect of Cobalt 60 Teletherapy. AECL's traditional superb and tested quality is maintained and, in fact, improved.

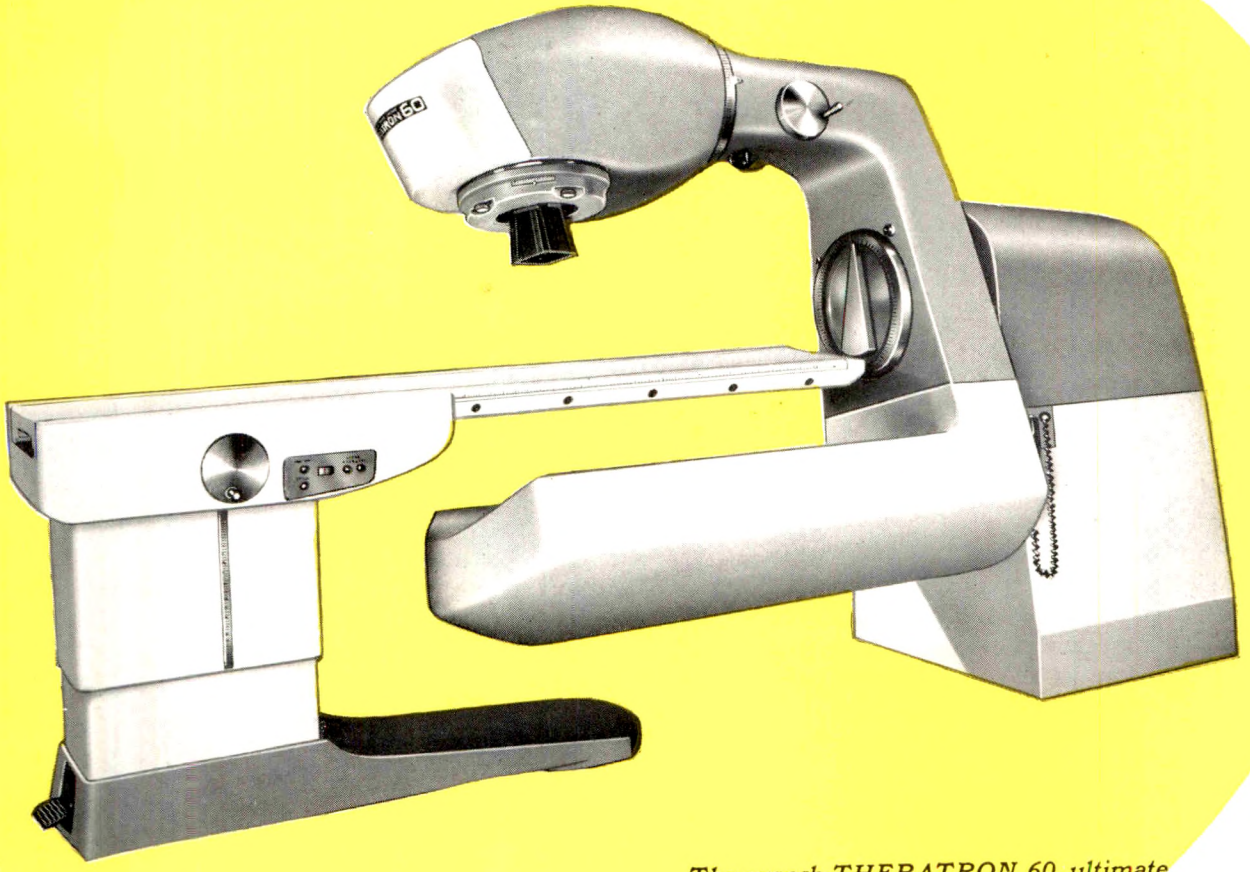
There are so many NEW features and advances that they cannot be listed here. These equipments should be explained fully and, if possible, seen and demonstrated to be fully appreciated.

AECL has representatives in every section of the world available at your convenience. If you cannot see the THERATRON 60 and the ELDORADO 6 on display at the International Congress of Radiology, please write to AECL and a representative will be glad to call at no obligation to you.

THE ELDORADO 6 offers all the outstanding features of the THERATRON 60 that are included in a stationary unit. Available with AECL's newly styled stretcher.

Cobalt 60 is here NOW!

THERATRON 60 and the

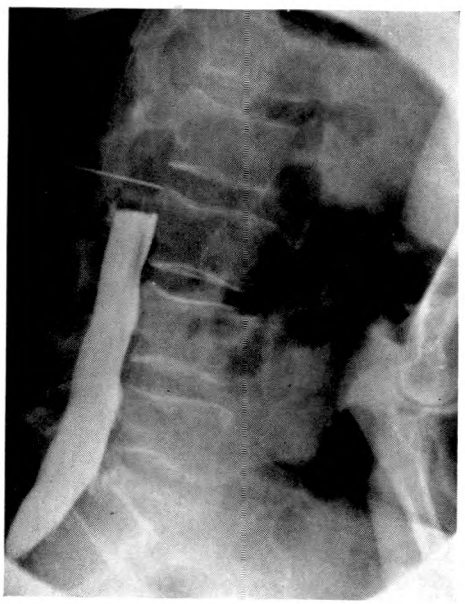


The superb THERATRON 60, ultimate in Cobalt 60 Teletherapy equipment embodying the most desirable features of the highest priced equipment, plus ALL AECL's NEW FEATURES.

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62-1M





Dynamic Myelography

These two radiographs of the same patient demonstrate the bulging of the annulus fibrosus during hyperextension and flexion, respectively, of the vertebral column. 30 cc of ***Pantopaque*** contrast medium was used. Note how this technique permits visualization of the posterior surface of the vertebral canal.

"PANTOPAQUE" *is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized by the Research Laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iophendylate Injection, U.S.P., by Lafayette Pharmacal Inc. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*

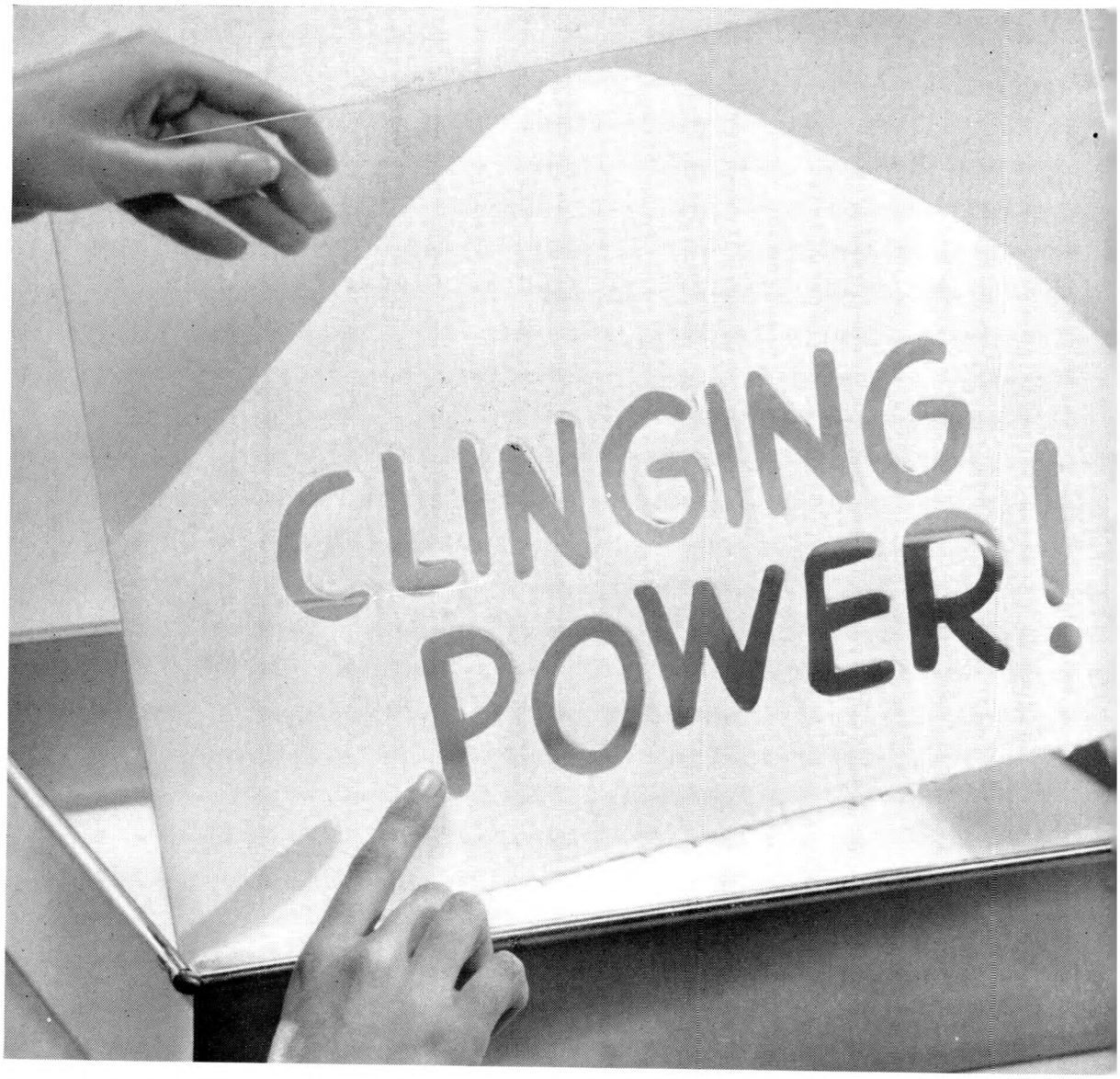
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COLLIMATOR



Four tiers of shutters, the uppermost close to the focal spot, ensure maximum interception of penumbra-causing off-focus radiation. Available as standard COLLIMATOR or as lower priced COLLIMATOR JUNIOR

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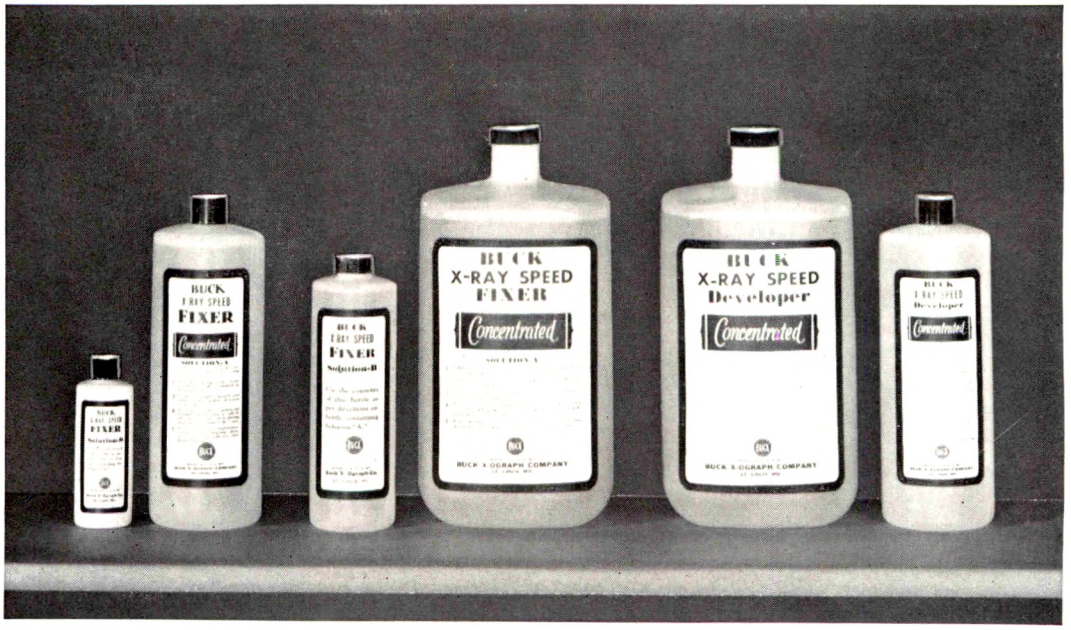


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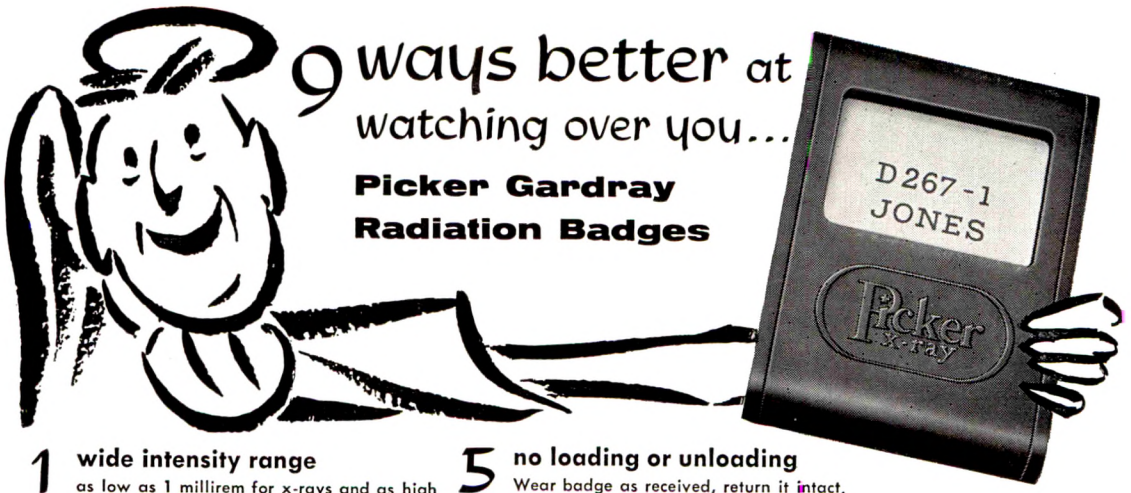
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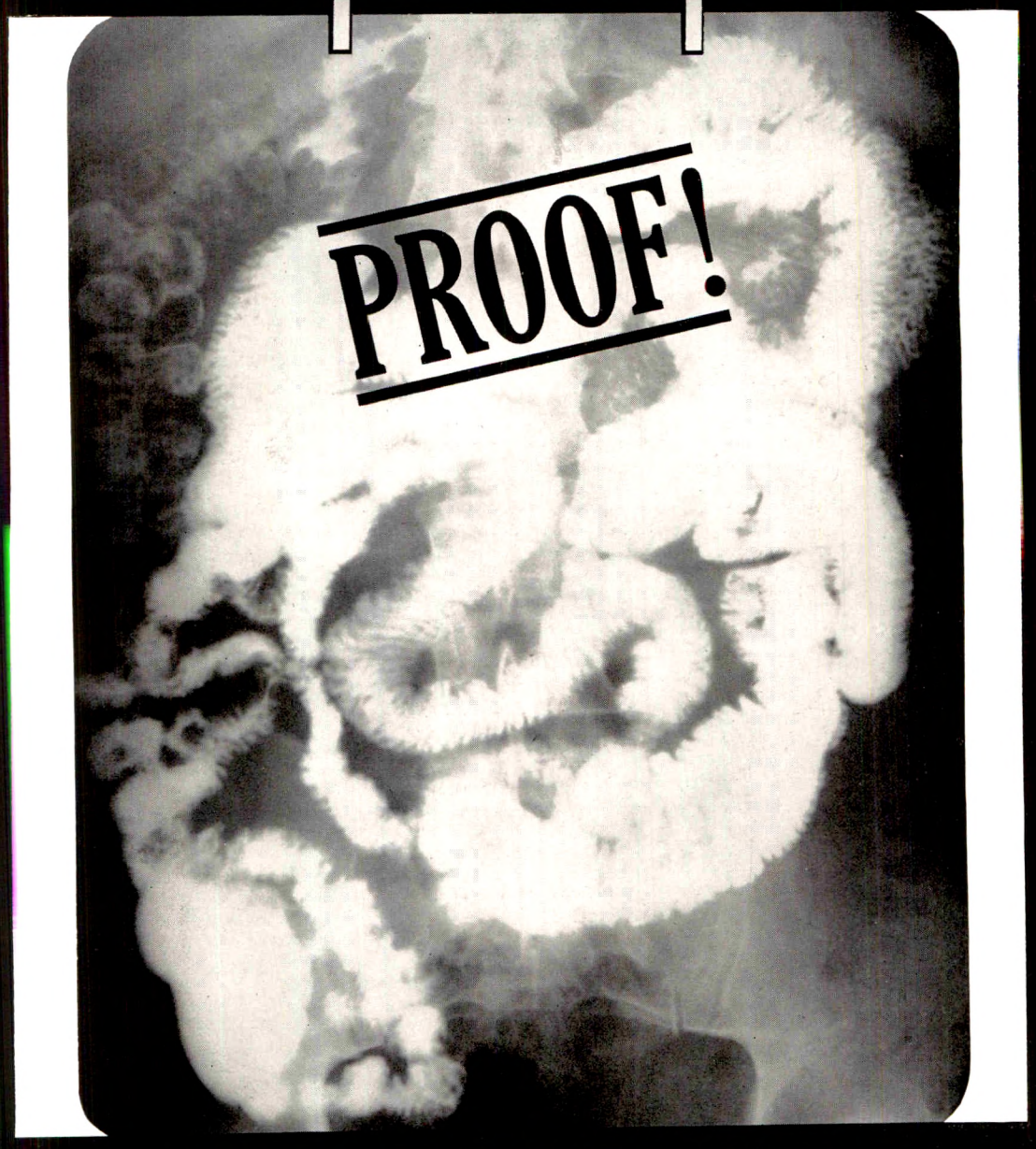
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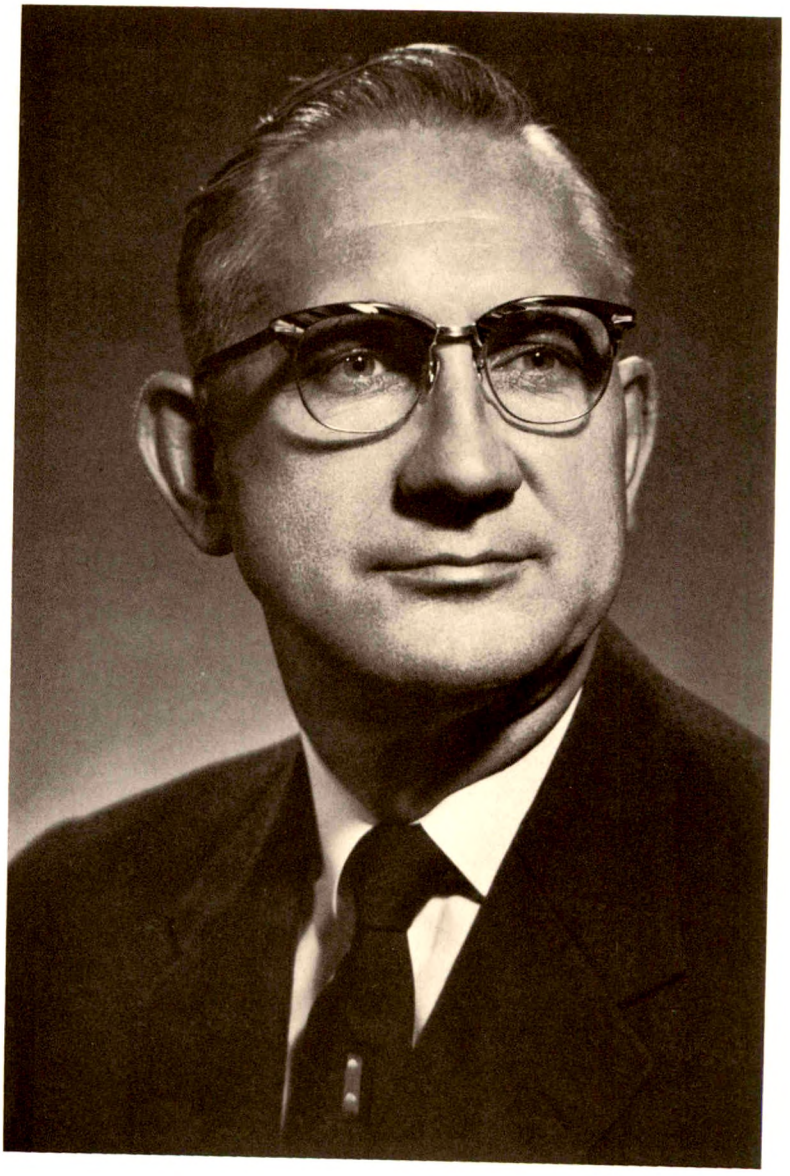
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HAROLD O. PETERSON

Caldwell Lecturer, 1961

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INTRODUCTION TO THE CALDWELL LECTURE, 1961

By J. P. MEDELMAN, M.D.
ST. PAUL, MINNESOTA

Doctor Leucutia, Doctor Peterson, Members and Guests of the American Roentgen Ray Society:

We are gathered this evening once again to freshen the memory of Eugene Wilson Caldwell by means of a lecture given in his name by a distinguished member of the present generation of scientists. To this end, since 1920, time has been set apart at the Annual Meeting for a formal lecture on a topic of particular interest.

Eugene Caldwell devoted most of his productive years to the furtherance of diagnostic roentgenology. He was born in Savannah, Missouri, in December of 1870 and remained there until his graduation from the high school. As a boy his interests were in mechanical things and scientific apparatus rather than in the usual children's pastimes. His innate predilections coincided with the early breakthroughs of the electrical marvels which we take as a matter of course today. When he was seventeen, he entered the University of Kansas to study electrical engineering. In his second year he became an assistant in the physics laboratory.

Each summer during his college career, he worked with Professor Blake of the Department of Physics on submarine telephonic communication. This work was done mainly off the New England coast and was successful. It led to employment on the Atlantic Seaboard in professional electrical work.

Late in 1895 he was in the employ of the New York Telephone Company. The scientific world was excited over the news of Röntgen's discovery. In 1897 Caldwell purchased an X-ray machine from a commercial photographer who had discovered early that roentgenology could not be exploited as a popular diversion. Late that year he opened what was probably the first X-ray office in New York City, prepared to do, according to his card, "X-ray work for physicians."

His name soon became well known in the progress of medical diagnosis by X-ray. He was invited to operate an X-ray laboratory for the University and Bellevue Hospital Medical College. This was established in 1909 as the Edward N. Gibbs Memorial Laboratory, and Caldwell directed it until 1908. He matriculated in the fall of 1902 at the school where he was teaching and was graduated with the degree of Doctor of Medicine in 1905.

Dr. Caldwell's contributions to radiology were of great importance, not only from the technical standpoint in which he was a genius, but from his concept of the dignity of the profession, and his concern for the fitness of the persons engaged in it. A glimpse into his character may be gained by quoting from an article on diagnosis which he published in 1908. He stated, "Such examinations should not, therefore, be undertaken lightly nor intrusted to careless or incompetent persons, not to those who have not the proper appreciation of the responsibility involved in deciding a question upon which may depend the health or even life of a fellow being."

He maintained a shop in which were made most of the instruments used in his office, from a simple tube-stand to a stereo-fluoroscope. He devised the Caldwell electrolytic interrupter, an induction coil, various types of X-ray tubes, a bedside set and a self-regulating valve tube. He is best remembered for his contributions to roentgen examination of the nasal accessory sinuses, and for a book which he wrote in collaboration with Dr. Pusey on "Roentgen Rays in Therapeutics and Diagnosis."

Dr. Caldwell served in World War I with the rank of major at the time of his death. He died on June 20, 1918, as the result of injuries received while experimenting with roentgen rays in the early days of his career.

The Caldwell Lecturer is chosen each year by the President-Elect of the Society. It is truly an honor for me to introduce this evening the Lecturer of 1961, Dr. Harold O. Peterson.

THE RADIOLOGIST AND THE SPECIAL PROCEDURES*

CALDWELL LECTURE, 1961

By HAROLD O. PETERSON, M.D.
MINNEAPOLIS, MINNESOTA

THERE are several signs which point to the development of a new, yet ill-defined specialty in the field of diagnostic roentgenology. The American Board of Radiology has recognized in recent years that certain important aspects of diagnostic radiology were not being included in their examinations. In an effort to at least partially remedy this situation, another diagnostic examination has been added which has quite logically and without any specific effort towards selecting a name become known as the *special procedures* examination. During this same period, in the radiology departments of the larger hospitals, particularly the teaching hospitals, *special procedures* have been developing very rapidly and have been occupying more and more of the radiologist's time. Concomitantly, on the programs of the local and national radiological societies' meetings, well exemplified by this meeting, there are increasing numbers of papers and exhibits and instructional courses dealing with *special procedures*. In view of this changing situation which affects most of us to some degree and the younger radiologists to a greater degree, a discussion concerning the radiologist and the special procedures would seem to be in order. The first part of the Lecture will deal with the entire subject of special procedures in a rather general manner, and the last part will cover in a more specific way some of my own experiences in two types of special procedures, myelography and discography.

THE SPECIAL PROCEDURES IN GENERAL

What is meant by the term *special procedures?* It is unlikely that this term, as applied to diagnostic radiology, has ever been defined. In trying to develop a definition, the conclusion is reached that it cannot be readily or simply described. At first consideration one might feel that only newer techniques developed in the last 10 to 15 years would be considered as *special procedures*. However, I believe everyone would agree that pneumoencephalography, first described 44 years ago, is still a *special procedure*. On the other hand, there are some comparatively old examinations, such as cholecystography and intravenous urography, which at the time of their inception might properly have been rated as *special procedures* but have become so commonplace in hospital as well as office practice that we now think of them as more or less routine procedures. Common usage, however, is not necessarily an indication that the examination has ceased to be a *special procedure*. Myelography and bronchography, first described in the early twenties, are now performed very frequently in many radiology departments and still are justly regarded as *special procedures*.

Most *special procedures* are concerned with the injection of some kind of contrast medium into the vascular system or into pre-existing body cavities. The Swedish investigators have been credited with the motto, "inject every body cavity with some type of contrast agent, and if there is no known body cavity, create one." Limiting the definition, however, to only those techniques requiring the injection of contrast media would not encompass all *special procedures* since such examinations as laminagraphy, kymography, magnification techniques, foreign body localization and perhaps even pelvimetry and mammography might well be classified as *special*

* Presented at the Sixty-second Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 26–29, 1961.

TABLE I

SPECIAL PROCEDURES
CLASSIFIED ALPHABETICALLY

1. Angiography
2. Arthrography
3. Bronchography
4. Cardiography
5. Cineradiography
6. Cystourethrography
7. Discography
8. Foreign Body Localization
9. Hysterosalpingography
10. Intraosseous Venography
11. Kymography
12. Laminagraphy
13. Lymphography
14. Magnification Technique
15. Mammography
16. Myelography
17. Pelvimetry
18. Percutaneous Transhepatic Cholangiography
19. Pneumoencephalography
20. Pneumomediastinum
21. Pneumoperitoneum
22. Renal Cyst Injection
23. Retroperitoneal Air Injection
24. Sialography
25. Splenoportography
26. Urography (retrograde)

procedures. The reason mammography could be included as a special procedure is the fact that even though it is not a new or difficult technique it is one with which most of us are not familiar, and we would not immediately feel qualified to supervise or interpret the examination.

Perhaps the most useful and understandable attempt at a definition would consist of an alphabetical classification of the various major groups of examinations that belong in this category such as shown in Table I. In some respects this classification is too simple, particularly in large areas as angiography and cardiography. Consequently, a much more elaborate classification (Table II) has been devised, based largely on various organ systems and to a lesser degree on different techniques. This is more inclusive and descriptive and possibly more useful. Still a third classification, which might also have some merit, could be based largely on the type of contrast media used, *i.e.*, water soluble or absorbable, oily or nonabsorbable, gas, or special techniques with no additional contrast agent (Table III). It is hoped that almost everything which might be considered as a significant *special procedure* today can be found in the aforementioned classifications. In the future some of these so-called *special procedures* may lose this admittedly artificial distinction and may then more properly belong in the category of routine examinations.

It is quite striking on reviewing Table II that the vast majority of *special procedures* do entail the injection of a contrast medium into some part of the body. This demands the services of a physician, even though the procedure might be as simple as injecting the veins of an extremity or as difficult as an intracardiac catheterization. It is in the performance of this technique that the question is properly raised, "What should the role of the radiologist be?"

It is my feeling that the radiologist should play a very active role in the performance of almost all of the procedures listed in these tables in addition to supervising the purely roentgen aspects of the technique. With two or three exceptions, which would include such things as ventriculography and retrograde pyelography, the radiologist could well be the person best qualified to master these techniques. Based on the belief that, "the one who can do the job best is the one who should do it," the radiologist is in an advantageous position to become the best since he is seeing cases from many different consultants and should, therefore, have a larger volume than any one of the referring physicians. This obviously will give him the opportunity for a large and continuing experience which is of prime importance if these procedures are to be expertly performed with the least discomfort to the patient. Furthermore, the radiologist should be the person most keenly desirous of having an excellent roentgen study and thus should have the greatest personal interest in controlling all of the technical maneuvers.

Finally, there are many procedures in which fluoroscopy plays either a major part or at least a supporting role. Some of these procedures are myelography, discography, cardiac catheterization, renal arteriography, renal cyst puncture and splenoportography. The radiologist, by taking advantage of his training in fluoroscopy, is often better equipped than anyone else to produce the finest results.

TABLE II

SPECIAL PROCEDURES
CLASSIFICATION BY ORGANS, SYSTEMS AND TECHNIQUES

I. *Circulatory System*
- A. Heart and Great Vessels
  - 1. Intravenous angiocardiography
  - 2. Right-sided catheterization and selective cardiography
    - (a) Wedge arteriogram
  - 3. Retrograde catheterization into left ventricle
  - 4. Transbronchial auricular puncture
  - 5. Transthoracic left auricular puncture
  - 6. Transthoracic left ventricular puncture
  - 7. Retrograde brachial or subclavian aortography
  - 8. Coronary artery catheterization
  - 9. Injection of root of aorta through catheter
    - (a) Cardiac arrest and Valsalva maneuver
  - 10. CO_2 injection of right auricle
- B. Lumbar Aortography
  - 1. Catheterization
  - 2. Direct needle puncture
  - 3. Intravenous aortography (with dextran)
- C. Renal Angiography
  - 1. Selective catheterization or ring catheterization
  - 2. Direct lumbar aorta puncture
  - 3. Intravenous aortography and nephrotomography
- D. Peripheral Arteriography (extremities)
- E. Peripheral Venography
- F. Venacavography
- G. Splenoportography
  Needle or catheter technique
- H. Hepatic Angiography
- I. Intraosseous Venography
- J. Lymphography

II. *Central Nervous System*
- A. Cerebral Pneumography
  - 1. Encephalography
  - 2. Ventriculography
- B. Cerebral Angiography
  - a. Arterial Injection
    - 1. Carotid puncture
    - 2. Vertebral puncture
    - 3. Subclavian puncture
    - 4. Retrograde brachial puncture
    - 5. Arterial catheterization via subclavian, brachial and femoral vein
  - b. Dural Sinus Venography
- C. Myelography
  - 1. Gas
  - 2. Water soluble opaque media
  - 3. Nonwater soluble opaque media (pantopaque, lipiodol, etc.)
- D. Discography

III. *Genitourinary Tract*
- A. Retrograde Urography
- B. Cystourethrography
- C. Seminal Vesiculography
- D. Retroperitoneal Air Studies
- E. Renal Cyst Injection
- F. Hysterosalpingography

IV. *Gastrointestinal Tract*
- A. Sialography
- B. Percutaneous Transhepatic Cholangiography

V. *Miscellaneous*
- A. Bronchography
- B. Pneumoperitoneum
- C. Pneumomediastinum
- D. Arthrography
- E. Thorotrast Studies of Liver and Spleen
- F. Retroperitoneal Air Studies

VI. *Special Techniques*
- A. Laminagraphy
- B. Autotomography
- C. Magnification
- D. Kymography
- E. Mammography
- F. Foreign Body Localization
- G. Pelvimetry
- H. Cineroentgenography

Viewing this matter from the standpoint of the general radiologist there are some disadvantages of having the radiologist so intimately involved with these techniques. The first disadvantage is the fact that a large amount of time is required. Most radiologists cannot devote this much time to these technical matters. However, since some *physician* must do this, perhaps it is just as logical that the radiologists find the time as it is for the surgeon or internist or neurologist.

Another deterrent is the fact that a certain amount of special training in these techniques is necessary. One cannot become a specialist of this type simply by proclamation. Unfortunately, there are not enough training centers available at this time which also have a sufficient volume of material to train all of the interested radiologists. We can nevertheless aim towards a gradual trend in this direction, and in the larger centers the radiologists can make every effort to become proficient in these tech-

nical skills and train their residents accordingly.

Another objection which has been voiced concerning the matter of having the radiologist do these procedures is that of the added hazard or risk assumed by the radiologist. This should be no greater for the qualified radiologist than for any other physician.

In the smaller hospitals the day may never come when it will be necessary or desirable to do all of these *special procedures*. However, if and when the need does arise to participate in any one of these more difficult techniques the radiologist should be in the forefront developing his skills, in conjunction with the clinicians if necessary, but at least taking an active part in any new project that needs to be developed. By so doing he will know as much or more than anyone else in the area and can continue to build on this background rather than slip further and further behind.

It is unlikely, however, that any one radiologist would become proficient in all of the aforementioned procedures even in the larger radiology departments with adequate material. In the first place, simply to decide which procedure and which technical approach would yield the most information might sometimes require an exhaustive knowledge of the pathologic anatomy and physiology in question. Likewise, a very complete understanding of the clinical problem would be essential, and it should not be expected that radiologists would have as much clinical judgment and diagnostic acumen in all fields as each clinician does in his own field, but he should at least be able to speak the same language. In the areas of cardiovascular disease and neurologic disorders, this is no small task. Consequently, as time goes on we will very likely find ourselves concentrating, as individuals, on certain types of *special procedures*. It should be entirely possible and perhaps desirable to continue to practice general radiology and at the same time develop a keen active interest in certain *special procedures*. Perhaps the day will come when as a result of long, continued,

TABLE III

SPECIAL PROCEDURES
CLASSIFICATION BY CONTRAST MEDIA

I. *Water Soluble or Absorbable Contrast Media*
- A. Cardiography (I.V.—Selective-Retrograde Catheter-Transthoracic)
- B. Aortography (Direct Puncture-Catheter-I.V.)
- C. Selective Arteriography (renal)
- D. Peripheral Arteriography (extremities)
- E. Cerebral Angiography
- F. Peripheral Venography
- G. Venacavography
- H. Intraosseous Venography
- I. Splenoportography
- J. Myelography
- K. Discography
- L. Cystourethrography (retrograde and voiding)
- M. Retrograde Urography
- N. Nephrotomography
- O. Seminal Vesiculography
- P. Renal Cyst Injection
- Q. Bronchography
- R. Arthrography
- S. Hysterosalpingography
- T. Lymphography
- U. Percutaneous Transhepatic Cholangiography

II. *Oily or Nonabsorbable Contrast Media*
- A. Bronchography
- B. Myelography
- C. Cystourethrography
- D. Seminal Vesiculography
- E. Hysterosalpingography
- F. Sialography
- G. Lymphography
- H. Thorotrast for Liver and Spleen

III. *Gas*
- A. Encephalography
- B. Ventriculography
- C. Myelography
- D. Arthrography
- E. Cystography
- F. Retroperitoneal
- G. Intraperitoneal
- H. Pneumomediastinum
- I. Cardiography
- J. Pyelography

IV. *Special Techniques* (*no contrast media added*)
- A. Laminagraphy
- B. Kymography
- C. Pelvimetry
- D. Foreign Body Localization
- E. Magnification
- F. Autotomography
- G. Mammography
- H. Cineroentgenography

extensive exposure to these *special procedures* we may feel as much at home with these examinations as we now do in studies of the gastrointestinal tract. At the present time, however, it is no more logical to expect any one radiologist to be expert in the doing and interpreting of all these procedures than it is to expect one surgeon to be expert in all phases of general surgery as well as cardiac surgery, neurosurgery, urologic surgery, orthopedic surgery, etc.

SOME PARTICULAR SPECIAL PROCEDURES

I should like to leave this part of the discussion which has dealt in a very general way with the over-all subject of *special procedures* and move into the comparatively specific field of neuroradiology, dealing with only a few aspects of myelography and discography. I believe most of us will agree that the entire field of neuroradiology is relatively difficult. Part of the reason for this continuing difficulty is the fact that we do not have sufficient repeated daily exposures to these examinations. Without this repeated practical contact, we cannot hope to become proficient in pneumoencephalography, cerebral angiography or even the interpretations of the minute subtle findings on plain skull roentgenograms. There are a mere handful of radiologists in this country who would claim competence in the entire field of neuroradiology and most likely this same situation obtains throughout the world. This is in sharp contrast to the thousands of radiologists who are quite capable and competent in the area of the gastrointestinal tract or chest.

There are few radiologists who have had as much experience, in terms of numbers of patients examined, in the area of myelography as they have had in gastrointestinal examinations. Nevertheless, a large number of papers have been written on myelography often based on a relatively small experience, particularly as compared to the usual experience in other fields. These same authors would not write papers on duodenal ulcers for example even though they undoubtedly have seen more duodenal ulcers than cord tumors or herniated discs. The obvious explanation is the fact that we tend to write about the things which are either new to us or concerning which we know very little.

The points to be emphasized in the field of myelography and discography are not new nor are they particularly erudite, but they are based on a long and active experience with these procedures. A detailed statistical analysis of the approximate 4,000 myelograms which are the basis of these observations might be interesting but would be almost impossible to obtain at this time. A comprehensive review of the entire subject of myelography and discography would be boring and out of place. Consequently, the discussion will be limited to a few selected impressions concerning myelography, discography and lumbar disc disease which have developed during the past 25 years and which perhaps merit further emphasis.

MYELOGRAPHY

It would seem unnecessary at this late date to make any reference to what constitutes good myelographic technique when using nonabsorbable media such as pantopaque or lipiodol. My observations, however, gained from talking with candidates who were being examined by the American Board of Radiology and from patients who have had myelograms elsewhere leads me to believe that there is much room for improvement in the manner in which myelography is often done. It is quite clear that there are only a few radiologists and very few training centers where the spinal puncture and the insertion and withdrawal of contrast media are done entirely by the radiologist. The inference is not that this is absolutely essential in order to obtain a proper examination in every case, but assuredly a better examination can be done more often by the well trained, interested radiologist than can be done by other specialists such as neurologists, neurosurgeons or orthopedic surgeons either alone or in conjunction with a radiologist.

By better examination I refer to: (1) the patient's comfort; (2) the proper injection and subsequent removal of the contrast medium; and (3) the roentgen technique and final diagnosis.

All the minute details of the correct technique of pantopaque myelography cannot be discussed here. In a general way, however, the major objectives should be as follows: (a) the procedure should be essentially painless; (b) the contrast medium should be injected correctly into the subarachnoid space; (c) a complete fluoroscopic and film record of the pertinent areas should be obtained; and (d) approximately 99 per cent or more of the medium should be removed 98 per cent of the time again without discomfort to the patient.

In order to obtain these objectives, the spinal puncture should be done on the fluoroscopic table with the patient prone and the puncture site selected fluoroscopically and marked on the skin with an appropriate ink or liquid lead pencil. The two best sites for puncture are between the second and third lumbar spinous processes or between the third and fourth lumbar spinous processes, midway between the two pedicles from side to side and not directly over any intervertebral disc. In order to accomplish this midline puncture, fluoroscopic control is essential. A satisfactory midline puncture cannot be routinely obtained with the patient lying on his side. A pillow or other padding under the abdomen is rarely necessary.

There are a few patients in whom myelography is desirable but who have such severe low back and leg pain that they cannot assume the prone position on the fluoroscopic table. In such cases it is permissible to do a lumbar puncture in the usual blind fashion with the patient lying on his side. If a suitable puncture is obtained, the injection of 50 to 100 mg. of procaine crystals dissolved in spinal fluid will completely relieve all their discomfort, and one can then complete the examination with the patient prone. The patients are extremely grateful at this juncture since this may be the first time they have been free of pain for weeks. Care must now be taken, however, to limit the examination to the lumbar region and to not inadvertently run the pantopaque and hence the spinal anesthesia up to the brain stem. These patients must also be watched very carefully as they are elevated towards an upright position since they may now have a sharp drop in blood pressure and become very faint in addition to the fact they now have no control of their legs as a result of the anesthesia.

Pantopaque is the medium of choice for the demonsrtation of all lesions from the sacral canal to the clivus. Lindgren[10] and others have advocated the use of air myelography for many lesions, but in our hands the results have not been good. The Swedish investigators[10] have also advocated abrodil, a water soluble medium, for examination of the lumbar region, but this requires the routine use of a spinal anesthetic, and I doubt that the results of this examination are significantly superior to pantopaque in the recognition of clinically symptomatic disc lesions to warrant the routine use of spinal anesthesia. The amount of pantopaque used should be rather large. Six cubic centimeters is the minimum for lumbar studies, and if the dural sac is wide in its transverse diameter, 12 to 18 cc. may be very advantageous, particularly in minimizing some of the nonsignificant deformities which are usually exaggerated by small amounts of medium (3 cc.). Nine to 12 cubic centimeters of pantopaque are recommended for all cervical studies. In some of the more difficult thoracic cases, still larger amounts of pantopaque are helpful. In one instance (Fig. 1) 56 cc. of pantopaque was used, filling the canal from S2 to C7 with the patient almost upright. This large amount did not cause any significant distress and over 99 per cent was removed. The usual indication for the use of such large quantities of pantopaque is to improve the study of the thoracic area, particularly in the presence of a kyphosis which makes it very difficult or impossible to pool small quantities of medium and

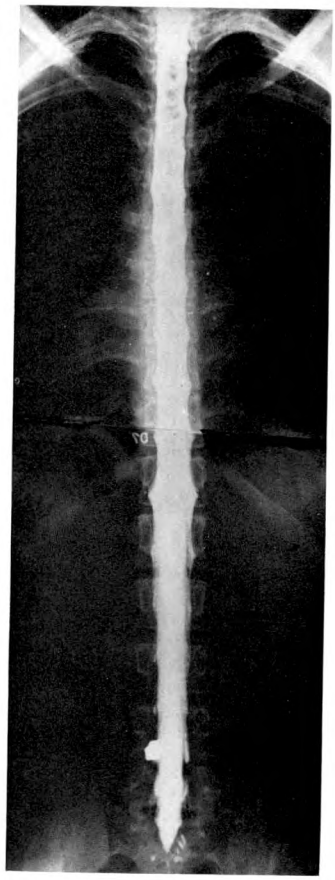


FIG. 1. Fifty-six cubic centimeters of pantopaque fills the subarachnoid space from sacrum to the seventh cervical vertebra. The illustration was prepared from two 14×17 inch films in order to demonstrate the entire length of the contrast column. An extensive angioma of the thoracic cord was convincingly demonstrated on spot roentgenograms as well as on conventional lateral roentgenograms.

thus adequately examine the region from about T5 to T10 with the patient prone. The oblique and lateral positions can often be employed to advantage in such situations, and on rare occasions the spinal puncture needle may have to be withdrawn and the patient placed in a supine position to permit good filling of the kyphotic segment.

It is my feeling that pantopaque should be removed from the subarachnoid space and that flooding of the cranial cavity should be avoided. It must be admitted that a fairly complete review of the literature to date contains very little documented evidence of harm resulting from residual pantopaque in the spinal canal. There are a few such reports in humans however,[3,4,5,13] and there is ample roentgen evidence that retained pantopaque may become fixed and immovable, indicating the development of some kind of adhesions or arachnoid proliferation.[15] This change has also been well identified in experimental animals.[1,2,7,16]

Three of our patients complained of extreme discomfort in the low back, perineum and legs especially when upright, even to the extent of being quite disabling, as the result of residual quantities of 1 cc. of pantopaque in one case, 5 cc. in another and 8 cc. in another. In the case with 8 cc., the medium had been present in the canal for almost 3 months and in the other 2 cases 6 weeks. On removing the greater part of this pantopaque (some had become fixed and not removable) most of these disagreeable and disabling symptoms disappeared. Most likely some, if not all, of the discomfort was due to the sheer weight or ballast effect of the pantopaque when the patients were upright.

It would be desirable to have a contrast medium somewhat less opaque than the presently available pantopaque. Perhaps this could be accomplished very simply by diluting pantopaque with some inert substance which would be miscible with pantopaque. This would be particularly desirable when very large quantities are to be used not only because the pantopaque column would be less dense but also because of the not inconsiderable expense associated with the use of these large amounts. If and when a water soluble, nonirritating, nontoxic, absorbable radiopaque medium is developed, it will certainly replace pantopaque and eliminate most of the refinements in technique so important today. The ultimate in contrast media for the subarachnoid space will have been obtained if an oral or intravenous route can be developed similar to what is now employed in oral cholecystography and intravenous urography. Until such a time, however, we must make the most of our presently available techniques.

An interesting and perhaps diagnostically significant phenomenon has been observed in 6 patients during pantopaque myelography. This consisted in the reproduction or aggravation of the patient's usual pain in the upper lumbar or thoracic levels when the pantopaque column was in contact with these segments. No significant myelographic deformities had been noted, and the aggravation of the pain ceased as soon as the pantopaque shifted away from the level in question. Some of these cases have been explored, and the surgeon has not found a gross lesion. More or less minimal signs of arachnoiditis were present. The patients have been relieved of their complaints of radicular pain, however, following rhizotomy at the appropriate levels. No explanation for this reaction is established as yet, but it is to be expected that more of these cases will be found and eventually the symptom complex will be better understood.

INTERVERTEBRAL DISC DISEASES

The remainder of the Lecture will deal with certain observations gained from the study of several thousand myelograms and many hundred discograms in patients suspected of having intervertebral disc disease in the lumbar area. Although these are not new ideas I believe they have been developed more or less independently and are based largely on clinical roentgenologic observations. They tend to agree with the much more basic anatomic studies done by many others dating back to Schmorl's[14] work in 1927, as well as with the more recent anatomic studies by Lindblom[8] (1944) and Harris and McNab[6] (1954) and with the impressions of several clinicians who have had large experiences with intervertebral disc disease. Rabinovitch[11] of Montreal has recently written a monograph on intervertebral disc disease in which he has combed the literature and made the interesting observation that in 1929, one year after Schmorl's paper, there were only 5 additional papers on disc disease. During 1935, one year after the highly significant clinical paper of Mixter and Barr, there were only 24 new publications. By 1959, however, 31 years after Schmorl's basic anatomic studies, 2,303 papers had been published dealing with intervertebral discs. Not many of the publications attempted to deal with the basic fundamental aspects of disc disease, and, as one might surmise, the underlying major cause of disc disease is still not determined or at least not agreed upon by the chief investigators. Although it is possible that clinically significant disc disease is due to a combination of several causes, it is also likely that there is a basic underlying common denominator for most cases whether it be degenerative, metabolic, congenital, developmental or chronic repeated trauma, and it would appear that very few cases are due solely to a specific injury. The monograph by Rabinovitch presents this aspect of the disc disease problem extremely well and poses many questions for future investigators.

It is probably unwise to enter into this discussion on etiology in view of the fact that my observations are largely roentgenologic with little in the way of basic anatomicopathologic studies. It is quite clear from these roentgen observations, however, that any explanation of the etiology of intervertebral disc disease must be compatible with the fact that most adults have some degree of anatomic abnormality in at least some of their discs. Indeed, it is difficult to find entirely normal lumbar discs in people over 35 years of age, and the older one gets the more likely it is that most of the intervertebral discs will be abnormal. This is not to say that everyone with abnormal intervertebral discs will have related symptoms. It would seem that anatomic pathology of the discs is extremely common, almost to the point of being universal in the older age groups, whereas symptomatic disc disease, while common enough, is comparatively rare. This is an extremely important concept to bear in mind when one is evaluating a myelogram or a discogram.

Myelographic deformities caused by bulging or posteriorly protruding discs are also very common, especially in the upper

lumbar and lower thoracic segments. These deformities are usually exaggerated if small quantities of contrast medium are used (less than 6 cc.). The real diagnostic problem is to try and determine which of these deformities are clinically significant. If the deformity observed myelographically is clearly incompatible with the clinical picture, it would seem quite obvious that such a myelographic appearance should be dismissed as far as explaining symptomatology is concerned. Quite often under such circumstances these myelographic deformities are bilateral, symmetric, rather rounded and smooth narrowings of the pantopaque column (Fig. 2, *A* and *B*). Such deformities when seen in the anteroposterior or oblique positions are usually associated with a slight generalized posterior elevation of the disc as determined in the lateral projection of the pantopaque column with the patient prone. More infrequently, this same bilateral symmetric narrowing will be due to a relative elevation of the discs with relation to a cupping of the posterior surface of the vertebral body with no actual posterior bulging of the disc being present (Fig. 3, *A*, *B* and *C*). Unilateral indentation into the dural sac in the upper lumbar area of similar smooth rounded nature must be viewed with more concern (Fig. 4); but again only if it agrees with the clinical picture should one seriously consider a symptomatically abnormal disc.

Frequently, at the L_4 level a somewhat different type of bilateral symmetric narrowing of the pantopaque column is seen often associated with an oblique radiolucent band visible in the lateral myelogram running anteriorly and caudally from the pos-

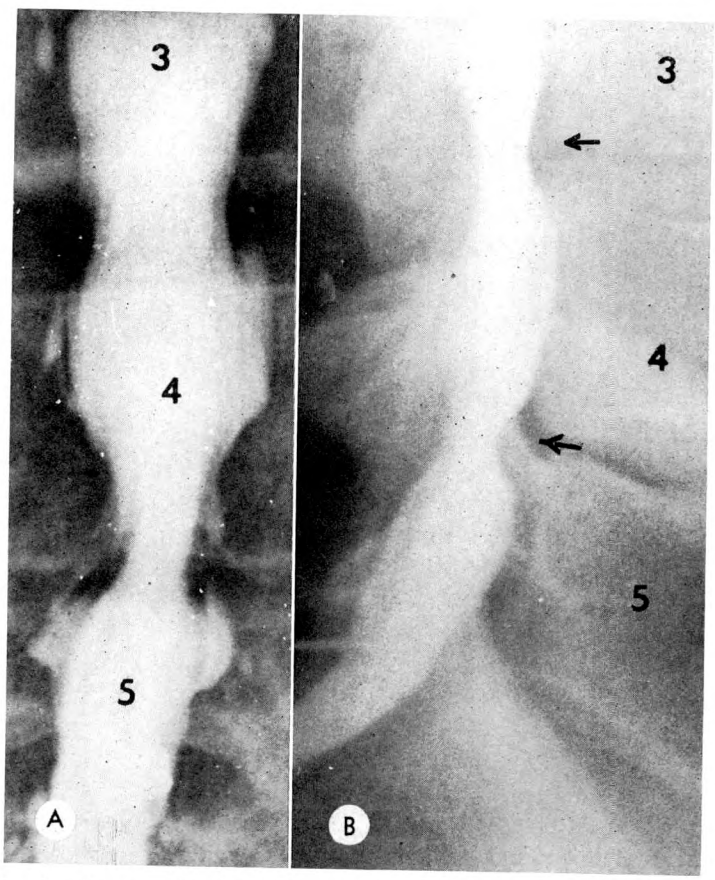


FIG. 2. (*A* and *B*) The discs between L_3 and L_4 and between L_4 and L_5 are bulging somewhat posteriorly as seen in the lateral view (*B*) and are producing a bilateral symmetric narrowing in the anteroposterior view (*A*). These were asymptomatic. There was a large intradural obstructing lipoma at T_{12}.

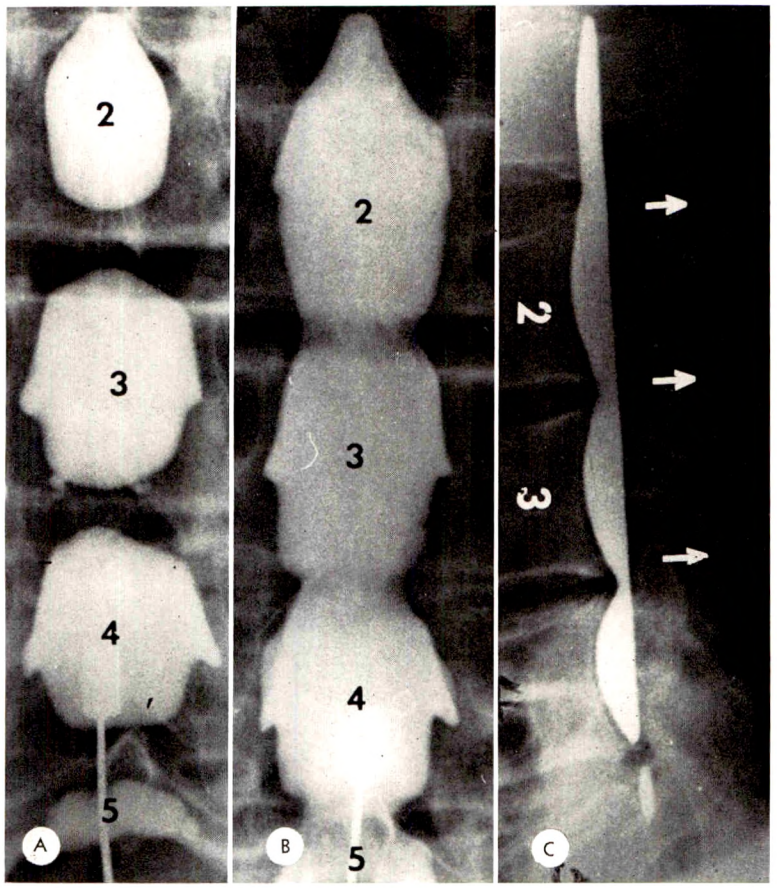


Fig. 3. (*A*) Anteroposterior view with 6 cc. of pantopaque suggests multiple bulging discs. (*B*) Only a slight bilateral symmetric constriction remains at each disc level when 12 cc. of pantopaque is used. (*C*) The lateral view indicates that these discs are not actually bulging posteriorly but are simply accentuated myelographically because of the cupping of the dorsal surfaces of the vertebral bodies.

terior aspect of the dural sac and ending near the level of the disc between L_4 and L_5 (Fig. 5, *A* and *B*). In the anteroposterior view at this same level there is an inverted V-shaped denser zone in the pantopaque column. Most likely this arrangement of the pantopaque is due to the normal course of the nerve roots within the dural sac extending from posterior to anterior and medial to lateral as they proceed distally. Exactly why this arrangement of the nerve roots is often associated with an asymptomatic bilateral symmetric narrowing of the pantopaque column at the L_4–L_5 disc level is not clear. Perhaps a very slight asymptomatic bulge of the disc in association with this arrangement of the nerve roots will combine to make this striking bilateral narrowing of the pantopaque column. Striking as this myelographic deformity is it has often been seen without appropriate clinical findings, and on some occasions the surgical findings have been correspondingly disappointing. If the lateral view clearly shows a large posteriorly bulging disc (Fig. 6, *A* and *B*), then this deformity will more likely be associated with symptoms. One should always be alerted by this appearance, however, and not go all out for a diagnosis of symptomatic herniated disc. The same inverted V can occasionally be seen at the L_3 and L_5 levels but is usually not as confusing as it is at L_4. On extremely rare occasions there can be two of these inverted V shadows present at adjacent levels.

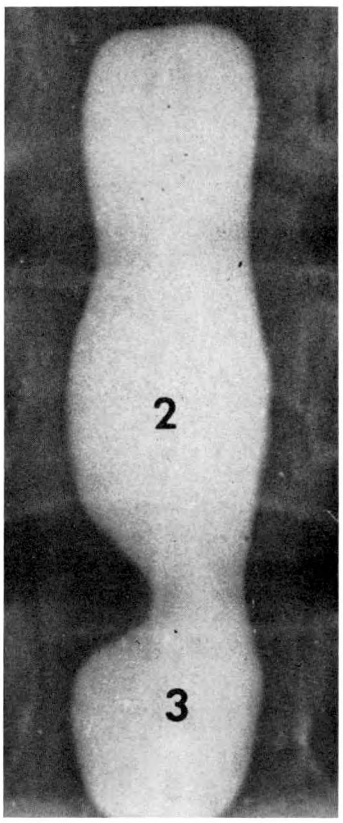


Fig. 4. Unilateral rounded type of indentation of pantopaque column in the upper lumbar levels is often not clinically significant even though it may be produced by a bulging disc. This patient had a meningioma in the mid-thoracic area.

We have recognized for many years a somewhat similar narrowing or even obstruction of the pantopaque column due to compression of the sac from behind (Fig. 7, *A* and *B*). The anteroposterior diameter of the bony canal is reduced, the laminae press in on the dural sac, may even overlap each other, and the articular facets crowd in from the sides. Verbiest[18] described this condition in 1954 and called it a developmental narrowing of the lumbar canal. This anatomic arrangement can sometimes cause enough compression of the dural sac to produce an obstruction myelographically which simulates the obstruction caused by a herniated disc. In lesser degrees it will resemble the bilateral symmetric narrowing described above. This posterior compression can also be associated with a bulging disc which then accentuates the obstruction. Since this condition may be present without symptoms or with atypical disc symptoms it is important to recognize the true anatomic nature of the process myelographically. This can only be done by means of the lateral projection which must show compression of the sac from the posterior aspect. When complicated by a bulging disc the sac is compressed from both anteriorly and posteriorly. Verbiest has stated that these patients have symptoms on standing and walking which disappear on rest and lying down. The most typical symptoms are tiredness and loss of power in the legs, feeling of numbness in sacral dermatomes and bilateral sciatica and lumbar pain. The surgeon should be forewarned so that an adequate decompression will be done at the proper level, and he will not be surprised in finding only a minor bulging or even normal disc.

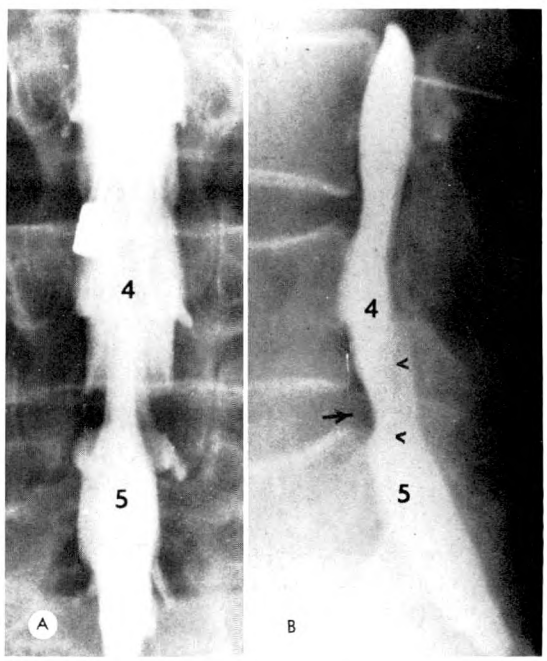


Fig. 5. (*A* and *B*) Bilateral notch type indentation which is a rather frequent finding especially at the L4-L5 level and often seen without any corresponding symptoms. This patient had a herniated cervical disc and no low back trouble. A slight bulge of the disc plus prominent nerve roots dividing in this inverted V manner probably accounts for this appearance.

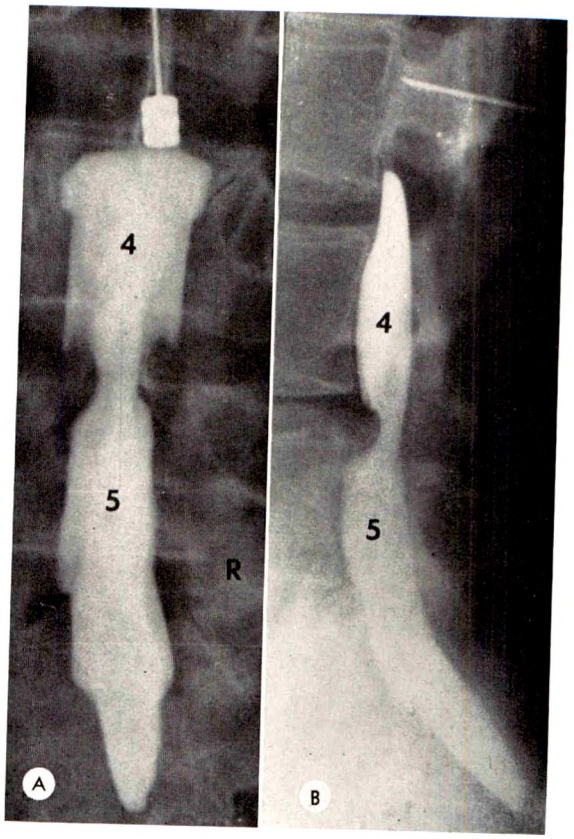


Fig. 6. Bilateral fairly symmetric deformity in anteroposterior view (*A*) at the L_4-L_5 level with a moderately large posterior bulge of the disc seen in the lateral view (*B*). The lateral view makes the findings seen on the anteroposterior view more significant. History of intermittent low back pain with radiation to both legs, mostly left, for 9 months. Rather large posterior bulging disc found surgically, more on left, removal of which produced complete relief of symptoms. Note the shallow indentation at L_5-S_1 on the right which was not clinically significant.

Since there are so many atypical types of deformities of the dural sac at all levels, it would be helpful if some roentgenologic criteria could be established which would clearly characterize the clinically significant discs. Unfortunately, this cannot be done with 100 per cent accuracy. As in many other types of diagnostic procedures there are a large number of myelograms which are quite clearly positive, and some are equally clearly negative. There is also a significantly large number of atypical myelograms which are not normal, but their clinical importance is not certain. If one

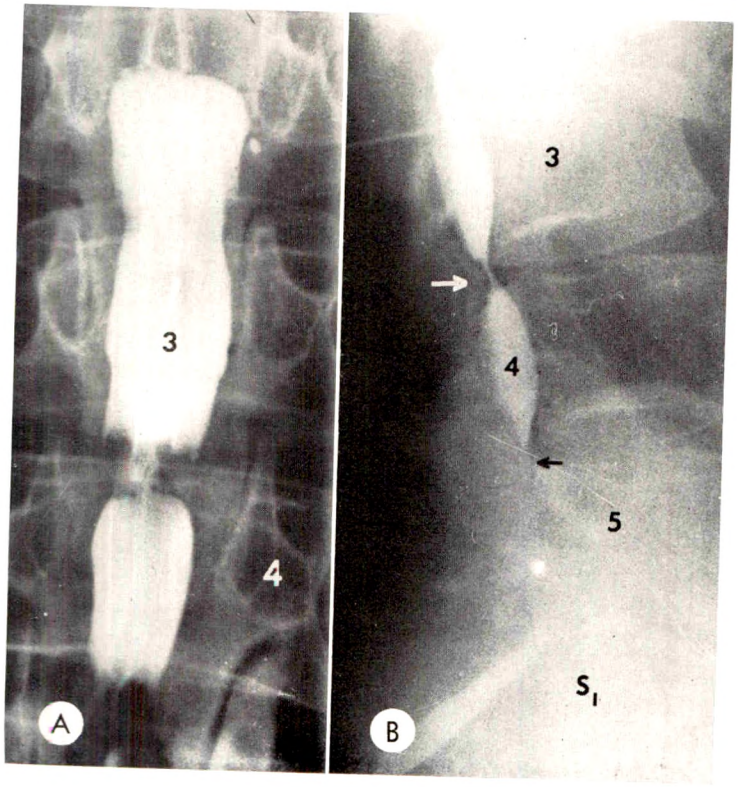


Fig. 7. (*A* and *B*) Partial block at L_3-L_4 (white arrow) and L_4-L_5 (black arrow) as a result of *posterior* compression of the dural sac. This might be due to a developmental narrowing of the bony spinal canal, thickening of the ligamentum flavum, and sometimes the block is further accentuated by a bulging disc.

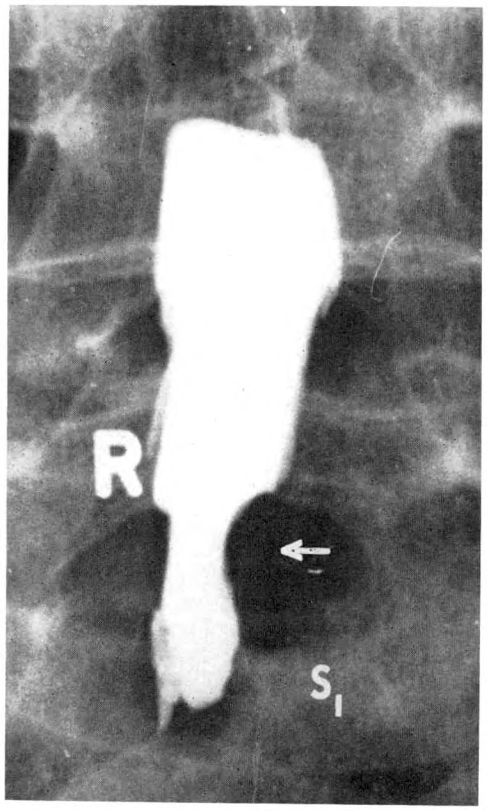


Fig. 8. Typical appearance of a herniated disc at the L5-S1 level (arrow). The deformity is unilateral, sharp and clean-cut, the axillary pouch is eliminated and the upper and lower contours of the indentation are not symmetric. This type of deformity will be associated with a typical herniated disc clinically and surgically almost 100 per cent of the time.

were allowed to select for comparative study only those cases in which he felt certain about the myelographic findings, the accuracy of the procedure would be very high. In this group the experienced myelographer would include primarily the unilateral indentations seen usually at the lower three lumbar levels. In addition to being unilateral, the indentation should be sharp and clean-cut, located directly opposite the disc level and preferably not symmetric as far as its upper and lower margins are concerned (Fig. 8). The upper end of the deformity should typically extend in a little further than the lower end. The indentation can be extremely small and still be significant, particularly at the L5 level. Minor changes in the appearance of the so-called axillary pouch are not in themselves accurate signs for a herniated disc (Fig. 9, *A* and *B*). When they are associated with an adjacent indentation into the main column of the dural sac, however, the appearance is often pathognomonic for a herniated disc (Fig. 10). As a rule the involvement of this axillary pouch occurs most often at the L5 level and is less frequently seen at higher levels because the pouch is often not located directly opposite the intervertebral disc especially above the L4 levels.

It is, of course, obvious that many clinically significant herniated discs will occur with myelograms which do not correspond to those described above. In some, the deformities will be unilateral but ragged, in others bilateral and asymmetric and in still others even bilateral and *symmetric*. The surgeon may find a definitely abnormal herniated disc in any of these situations, removal of which will alleviate the patient's complaints. Nevertheless, the more atypical the myelogram the more disappointing will be the surgical findings and the results. Similarly, there will be a higher incidence of poor correlation between the clinical and myelographic findings if the myelographic deformities are atypical.

It is unfortunate that there is no universally accepted and final method of verifying the accuracy of the myelographic findings in all disc cases even when surgery is employed. This is due to the fact that there is no clear-cut end point or microscopic technique which will produce a definitive answer such as one can obtain for example in carcinoma of the stomach. Thus it is almost impossible to arrive at any figure which will indicate the over-all accuracy of myelography in the diagnosis of herniated disc. Until better criteria are available for establishing the final diagnosis, we must depend not only on the surgeon's operative findings but also on the subsequent long term response of the patient to the surgical procedure. In order for such a study to be of

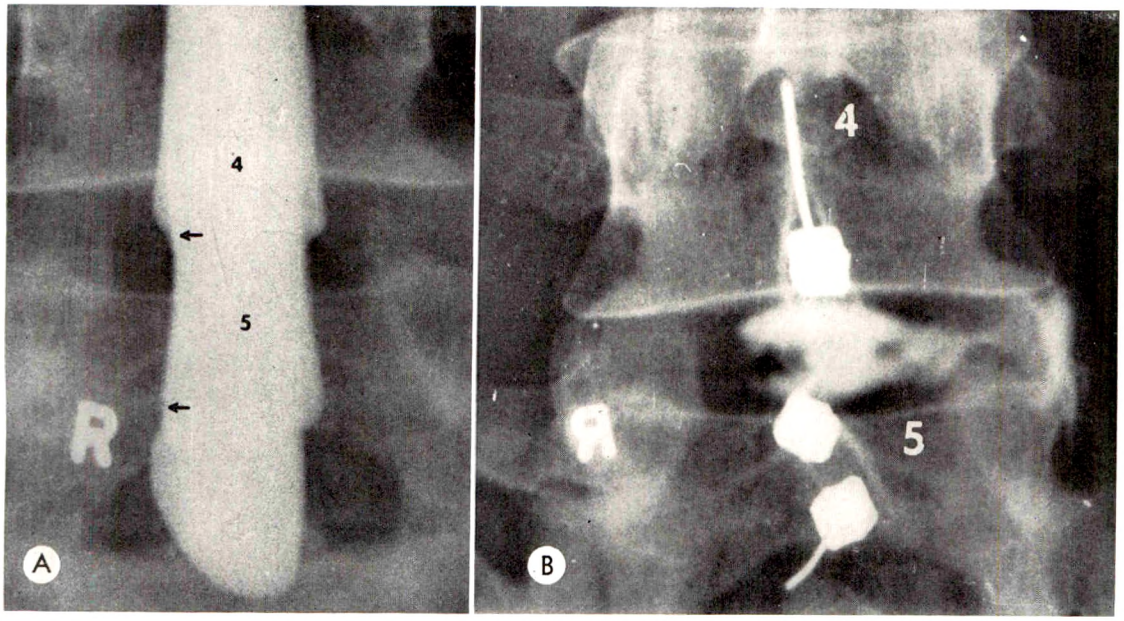


Fig. 9. (*A*) Normal myelogram of L4-L5. (*B*) Positive discography of L4-L5 with reproduction of patient's pain. Good surgical evidence for a herniated disc with good postoperative result. Note the slightly elevated axillary pouch L4-L5 and the absent pouch L5-S1 on right. Neither of these were significant.

value, all operative diagnoses should be made by an extremely competent surgeon with a great deal of experience in this field who would report his findings with meticulous accuracy. A postoperative follow-up for a minimum period of perhaps one year should be done in a like manner. Analyzing the myelogram in the light of this total picture would come closer to arriving at the accuracy of the myelographic diagnosis in the operated cases than anything so far reported.

The accuracy of a negative myelogram is almost impossible to decide since most of the patients are not explored. Some myelograms are more negative than others, or perhaps it would be better to say more normal than others. Quite commonly the distal end of the dural sac at the L5 and S1 levels is normally situated far posteriorly. This will permit a posterior herniation to be present without causing any myelographic

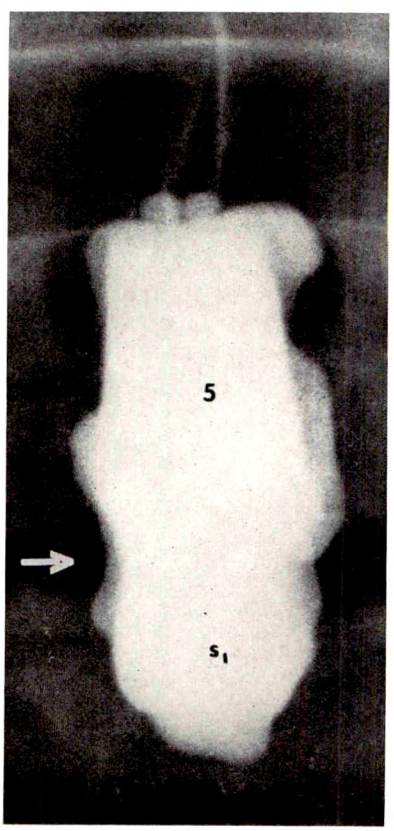


Fig. 10. Herniated disc at L5-S1 (arrow), with a shallow, slightly shaded indentation into the dural sac as well as a partial obliteration and elevation of the axillary pouch for the first left sacral root.

deformity. In other cases the transverse diameter of the dural sac, especially at the L5–S1 level, is quite small, again allowing for the presence of a posterolateral herniated disc without evidence of a myelographic deformity. Both of these situations can occur in the same patient, thus increasing the chances for a normal myelogram in the presence of a herniated disc. Conversely, the most accurate normal myelogram would result when the dural sac is quite wide and is located immediately adjacent to the bodies of the lumbar and sacral vertebrae. In such cases the accuracy of a normal myelogram may be as high as 90–95 per cent.

In light of the foregoing remarks which indicate that a herniated lumbar disc cannot always be completely ruled in or out with certainty by means of myelography, the question properly arises as to the indications for myelography. Many experienced surgeons and radiologists believe that myelography is indicated in all patients with suspected herniated disc in whom surgery is contemplated. There are, in addition, many other cases in which the clinical diagnosis is so uncertain that a final decision as to treatment can only be made following myelography. Perhaps the major advantage of routine myelography in all suspected disc cases is the fact that the spinal canal can be visualized in a manner accurate enough to significantly assist in making major decisions for either surgical or conservative management of the case To deserve this consideration, myelography must be performed with extreme care so as to provide the best myelograms, the best interpretations and in order to assure the patient and the attending physician that this study can be done without any real discomfort or risk to the patient.

DISCOGRAPHY

This technique described by E. Lindgren in the early forties and later developed by Lindblom[9] in the late forties has not become widely used. Except for a few centers which endorse it enthusiastically, it has largely been abandoned. The reasons for the failure of discography to gain widespread popularity are probably two in number: (1) discography is difficult to do; and (2) discography is difficult to interpret. The latter is the problem which is most distressing.

One would probably overlook the technical difficulties if a high degree of diagnostic accuracy was usually obtainable. Since this does not seem to be the case, the procedure has been criticized perhaps more than is justified. The difficulty in interpretation, as in myelography, is due to the fact that there are so many anatomically abnormal discs but so few causing symptoms. Discography does not offer an absolutely certain way of distinguishing between them.

There are 3 general areas where discography has proven useful: (1) in the occasional case of suspected intervertebral disc disease which cannot be diagnosed even after careful complete clinical and roentgenologic studies, it is sometimes possible to procure convincing evidence by discography either that the disc is normal or that it is not only anatomically abnormal but that the abnormality is clinically significant (Fig. 9, *A* and *B*); (2) there are, in addition, some rare instances of very atypical perineal, inguinal and abdominal symptoms which are due to disc disease and have been diagnosed by discography and relieved by disc surgery; and (3) much pathologic anatomy of the interior of the disc has been revealed by discography which cannot be readily obtained by other methods. Further anatomic correlation studies certainly should be carried out at necropsy, but the study of *in vivo* discograms to date has definitely broadened our concept of the intervertebral disc. Some of the pathologic anatomic changes which can be demonstrated by this method are Schmorl's nodules, intracorporal disc herniations anteriorly, tears in the cartilaginous plates, and holes in the posterior longitudinal ligament, in addition to the many degenerative changes within the discs themselves.

Another aspect of discography which is very important diagnostically is the production of pain on injecting water soluble contrast medium into the disc. Only the abnormal discs are painful when injected. The mechanism of this pain production is not clear. One explanation which has been suggested is that of simple distention of the disc with mechanical pressure on the nerve roots. This seems unlikely, however, since we have observed cases where only 0.1 cc. of medium has caused excruciating back pain. We have also injected in a few cases 1.5 cc. to 2 cc. of normal saline without pain and have then produced pain by injecting the same disc with contrast medium. We have also been able to completely relieve the severe pain which follows contrast injection by putting 2 cc. of 2 per cent procaine into the disc. Following the procaine it is possible to further distend the disc with more contrast medium without pain. These findings suggest the presence of sensory nerve endings within the annulus and probably in the posterior longitudinal ligament which are irritated directly by the hypertonic solution of contrast media and can be anesthetized by direct contact with procaine. Such sensory nerve fibers were first described by von Luschka in 1858 as the nervus sinuvertebralis and redescribed by Roofe[12] in 1940 as the recurrent lumbar nerve. This small root returns to the spinal canal from the lumbar root and extends downwards two or three vertebral segments, supplying the posterior longitudinal ligament and the annulus. This innervation might set up a reflex arc which could explain the occasional case in which inguinal or abdominal pain appears to be related to disc disease in the lower lumbar levels. The visceral afferent sensory fibers of the sympathetic chain in the abdomen might also be affected by abnormalities of the discs since they lie in contact with the vertebra anteriorly.

From a practical standpoint, our present attitude towards discography is to use it as a last resort in the occasional case of possible lumbar intervertebral disc disease where everything else has failed. This usually means that the clinical story and findings are atypical, the myelogram is either normal or atypical and the patient continues to have symptoms in spite of conservative treatment. Not infrequently discography in such a case will produce evidence which will either absolve the lumbar discs or incriminate one or more discs sufficiently to warrant disc surgery.

SUMMARY

A large number of *special procedures* are now being developed and used in many institutions. The radiologist of the future should strive to master these techniques as they are needed in his own area. This will require a certain degree of manual dexterity and expertness, particularly in the use of needles and catheters, and a special up-to-date knowledge of the various contrast media and injecting methods. The interested radiologist is in a unique position to become the most proficient in these procedures since he usually would have access to the greatest number of referred patients and hence a greater opportunity to develop his skills. Such practices have long since been established in some countries, particularly Sweden and Norway.

This has certainly been the case at the University of Minnesota and in all the hospitals in St. Paul as far as myelography and discography are concerned. All myelographies in these areas have been performed solely by radiologists for the past 20 years, and all discographies likewise for the past 10 years. This has clearly led to refinements in technique, has added to the patients' comfort and improved the diagnoses.

The acceptance of this total responsibility for the entire procedure by the radiologist has been gratefully acknowledged by the neurologists, neurosurgeons and orthopedic surgeons. This is supporting evidence that the expert services of the well-trained radiologist would be well received in many of the other *special procedures* as indeed is proving to be the case at the present time. The stature of radiology is thus enhanced,

the quality of the entire procedure is improved and, what is most important, the patient benefits from the added comfort and increasing accuracy in diagnosis.

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A TECHNIQUE FOR AIR ENCEPHALOGRAPHY USING A STRETCHER WITH A DETACHABLE SUPPORT FOR SITTING POSITION*

By MICHAEL SCOTT, M.D.
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A TECHNIQUE for partial or total air encephalography under local or general anesthesia, utilizing a standard hospital stretcher and modifying it with adjustable and removable supports (Fig. 1) is presented. The entire procedure can be performed without moving the patient from the stretcher.

TECHNIQUE

With this method a preliminary spinal puncture under local anesthesia can be made in the lateral prone position for an accurate spinal fluid pressure, thus giving the operator an opportunity to cancel the procedure if he considers the pressure a contraindication (Fig. 2).* If the pressure is below 200 mm. of water, the patient can be slowly raised to the sitting position at one end of the stretcher and the support for the upright position can easily be attached to the stretcher frame.† This support prevents the anesthetized or weak patient from slumping or falling and is reassuring and comfortable to the awake patient. If he should faint or go into shock, or have any untoward reaction at any stage during the procedure, the airplane seat belt lock can be instantly released, one vertical support quickly removed, and the patient eased to the lateral prone position on the stretcher, with the spinal needle still in place. The situation can then be evaluated and depending upon circumstances the procedure is either cancelled or resumed.



FIG. 1. A standard hospital stretcher modified with adjustable and removable supports for the sitting position.

If the patient has tolerated the upright posture, the stretcher is wheeled against the Franklin head roentgen-ray apparatus, the head is appropriately positioned against the cassette holder, depending upon whether the operator wishes to visualize the ventricular system or the basilar cisterna, and 10–15 cc. of air is slowly injected in approximately 10 seconds. Ten seconds after the air is injected, a pair of stereoscopic posteroanterior exposures are made (Fig. 3). The patient is adequately and comfortably supported by the upright

* We believe that any patient with a choked disk or a spinal fluid pressure above 200 mm. of water is a potential candidate for cerebral or cerebellar herniation. Such cases should have ventriculography if air studies are necessary. If visualization is desired of the infratentorial structures, a catheter cannula should be inserted into the ventricle and the latter decompressed before the air injection is made from below.

† Designed and constructed by Mr. Charles Thur of the Medical Center machine shop.

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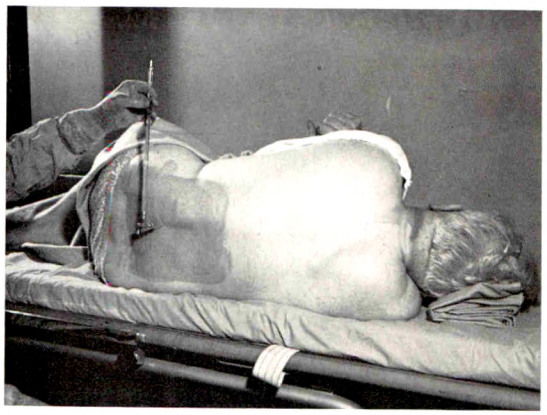


Fig. 2. Spinal puncture under local anesthesia permits accurate evaluation of the cerebrospinal fluid pressure and gives operator opportunity to cancel the procedure if he believes the pressure contraindicates air encephalography.

holder as well as the head holder during the 4 minute period necessary for the developing of the film. If adequate filling has been accomplished, the head is released from the head holder and held by an assistant in the appropriate position, and the stretcher is turned 90 degrees so that the head is presented in the lateral position to the cassette. Again a pair of stereoscopic exposures are made. Following this study the head is released and a lateral autotomogram is made. Films are developed, and, if technically satisfactory, the patient is slowly eased to the prone position on the stretcher and then wheeled to his room.

If visualization of the basal cisterna is desired, the patient is first placed in the lateral sitting position with the head partially extended and 10–15 cc. of air is injected, after which a pair of stereoscopic roentgenograms are taken. Following this an autotomogram and posteroanterior roentgenograms may be taken, depending upon the examiner's desires.

Visualization of the Lateral Ventricle in the Upright Position

This is done by extending the head and permitting the air trapped in the fourth ventricle, aqueduct and third ventricle to enter the lateral ventricles. If scout roentgenograms show inadequate filling, an additional 15–30 cc. of air can be injected with the head moderately flexed to permit the air to enter the ventricles. The head is then extended to get all the air into the lateral ventricles, and positioned for appropriate posteroanterior and lateral views.

Visualization in the Recumbent Position

If the ventricular filling is adequate, the needle is removed and the patient is gently lowered to the supine position on the stretcher. A pair of stereoscopic anteroposterior roentgenograms are taken with a vertical ray followed by stereoscopic roentgenograms with a horizontal ray (Fig. 4). The patient is then placed in the right lateral prone position and stereoscopic roentgenograms are taken with a vertical ray; roentgenograms in the same view are repeated with the patient in the left lateral prone position. The patient is finally placed in a face down position and stereoscopic roentgenograms are taken with a vertical ray followed by stereoscopic roentgenograms with a horizontal ray. If the roentgenograms are satisfactory, the patient is returned to his room on the stretcher.

Summary

A technique for partial or total air encephalography under local or general

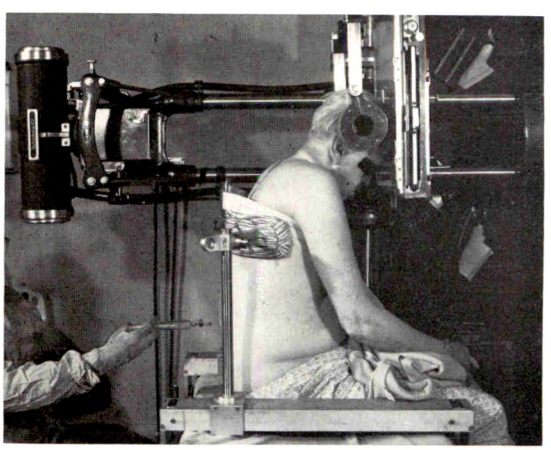


Fig. 3. Patient in position for posteroanterior view following injection of 10–15 cc. of air for visualization of the infratentorial structures, aqueduct and third ventricle.

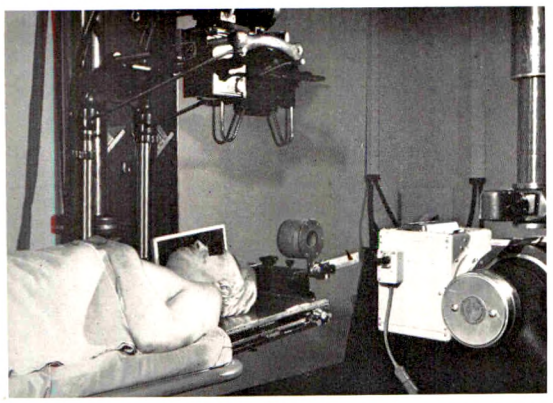


Fig. 4. Patient in position for supine anteroposterior view with a vertical ray. This is followed by a lateral view with a horizontal ray.

anesthesia is presented, utilizing the standard hospital stretcher, which has been modified with adjustable and removable supports for the sitting position. This permits total study of the patient in the sitting and/or recumbent position, without removing or lifting the patient from the stretcher. In résumé, it permits:

1. Preliminary spinal puncture in the lateral prone position enabling the operator to cancel the procedure if the pressure is above the acceptable levels.
2. Firm support of the patient during the study in the sitting position.
3. Movement of the patient to all necessary positions without removing him from the stretcher.
4. The immediate return of the patient to the lateral prone position if the procedure must be interrupted temporarily or permanently for any reason.
5. The return to the sitting position with the continuation of the procedure if the operator believes this indicated.
6. Total encephalography in the recumbent position following any studies desired in the sitting position.
7. The return of the patient to his room on the stretcher.

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OCCLUSION OF THE MIDDLE CEREBRAL ARTERY WITH RECANALIZATION*

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ENCEPHALOMALACIA is occasionally found in the presence of intact regional vessels. This has given rise to the hypothesis that vasospasm may be an underlying cause of cerebral infarction. However, it is also possible that thrombi or emboli occluding vessels may dissolve after producing infarction or that the vessel may recanalize. The following case is presented as an illustration of recanalization of a major cerebral blood vessel.

REPORT OF A CASE

H. H., a 55 year old woman, was admitted to the Kings County Hospital on September 29, 1959 with the chief complaint of right-sided weakness which came on after a fainting spell 5 days earlier. There was no history of hypertension, heart disease, or previous neurologic disorders. Physical examination revealed depression of the right corneal reflex, right central facial paresis, mild right hemihypalgesia and hemiparesis, and both motor and sensory aphasia with perseverations. Lumbar puncture disclosed no abnormalities. Left carotid angiography performed on October 16, 1959 showed complete occlusion of the middle cerebral artery as this vessel entered the sylvian fissure (Fig. 1). In the later films the terminal branches of the middle cerebral artery were opacified by way of anastomoses with branches of the anterior cerebral artery. Angiography was repeated on November 5, 1959 revealing an apparently normal middle cerebral artery with no evidence of occlusion (Fig. 2). In the interval between the two examinations the patient's neurologic symptoms remained relatively unchanged. Her aphasia became slightly less marked but was still present. By May 25, 1960 she had developed a Parkinsonian state, both in facial expression and tremors of the extremities. She was transferred to a state hospital on July 26, 1960, unimproved.



FIG. 1. Left carotid angiogram on October 16, 1959 demonstrating a complete occlusion of the middle cerebral artery. Later angiograms in the series showed opacification in the distal branches of the middle cerebral artery by way of anastomoses with branches of the anterior cerebral artery.

DISCUSSION

Lehrer[4] has reported a similar case. His patient was a 45 year old male who, following 12 hours of premonitory symptoms, suddenly developed left homonomous hemianopsia, left hemiplegia and hemianesthesia. Right carotid angiography performed on the third day revealed only opacification of the anterior cerebral artery in the early phases. In later phases branches of the middle cerebral artery were seen to fill in a retrograde manner. The anterior cerebral artery was shifted 1 cm. across the mid-line, probably because of edema. Three and one-half weeks later a second angiography was performed which was normal in

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all respects. During this interval there was progressive clearing of the sensorium and the hemianopsia as well as partial return of motor function.

When occlusion of the middle cerebral artery is demonstrated angiographically, a common finding is retrograde filling of peripheral branches of this vessel by way of anastomoses with branches of the anterior and posterior cerebral arteries.[3,7,8,9,12] Welch *et al.*[13] demonstrated the presence of collateral vessels 2 days following the onset of paralysis. Meyer[5] and Meyer and Denny-Brown,[6] working with monkeys, list as possible important factors influencing the formation of collateral circulation local changes in oxygen availability, temperature and pH but find the most important factor to be a reduction in the local head of pressure resulting in vasodilatation. Such changes in blood flow, according to these authors, are rapid and occur within 30 seconds after occlusion.

Although there is general agreement as to the existence of these anastomoses, there is considerable dispute about their function in limiting cerebral anoxia and in contributing to the alleviation of symptoms. It has been stated[2,9] that the collateral circulation bears no relation to clinical progress. At the other extreme, Solnitsky[10] believes that anastomoses play an important role in limiting the extent of infarction in the area of distribution of an occluded vessel. In areas of poor anastomotic development, he has found the area of infarction to be correspondingly greater. Gros *et al.*[1] agree that any improvement following cerebral artery occlusion must be due to anastomoses. They do not believe that recanalization of the occluded vessel can contribute to improvement in symptoms because of its late appearance, although such recanalization has been observed anatomically. The present case would tend to support the statements of these latter authors.

Taveras and Poser[11] have reported the angiographic findings in several children with cerebral hemiatrophy and infantile hemiplegia. They found a diminution in caliber and number of the middle cerebral branches. They felt that the smaller caliber suggested that vascular injury or actual thrombosis had taken place at an earlier age and had been followed by recanalization.

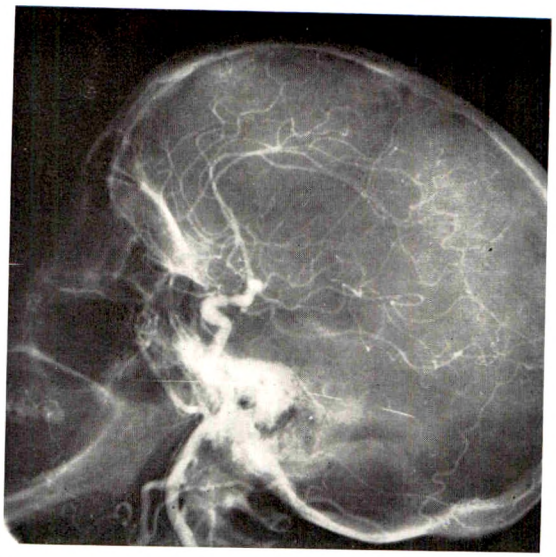


Fig. 2. Repeat angiogram on November 5, 1959. At this time there is no evidence of occlusion or stenosis of the middle cerebral artery.

SUMMARY

A case of angiographically demonstrated recanalization of the middle cerebral artery is reported. Reference is made to a similar case in the literature. The physiologic significance of this recanalization is open to question, as is that of leptomeningeal anastomoses following arterial occlusion. In the case reported, despite the presence of anastomoses and recanalization, there was no significant clinical improvement.

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FLOW GUIDED CARDIAC CATHETERIZATION*

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DURING the time required to read this sentence, the reader's antecubital veins will be traversed by 3 or 4 billion red blood cells, virtually all of which will, within 2 to 4 seconds, reach the pulmonary artery. The free phase of pulmonary embolization is similarly brief. Why, then, does it require an average of 10 to 30 minutes to pass a cardiac catheter into the pulmonary arteries? Explanation, if not justification, lies in the rigid catheter used for conventional venous catheterization. This report describes the use of highly flexible catheters, introduced percutaneously, for flow guide catheterization of the right heart and pulmonary vessels.

A 5 year search for catheter flexibility has led to success with rubber in several forms, including specially dipped latex tubing and military surplus electronic insulation. Silastic, an inorganic silicone rubber, proved to be the ideal material for our flexible tubing (Silastic X-30146, .08″ o.d. by .04″ i.d.). This tubing, percutaneously introduced into an adequate, unoccluded antecubital vein through a 12 gauge Robb-Steinberg angiocardiography needle, will, when advanced, pass with the aid of blood flow to and through the right heart chambers and into the pulmonary artery. Introduction through small, tortuous arm veins and those occluded by cutdown or narrowed by trauma is likely to be difficult or impossible. The larger the vein and straighter its course into the mediastinum, the less the likelihood that the tubing will pack up within the vessel instead of traversing it. The occasional tendency of the limp tubing to curl up rather than pass through the needle into the vein can be overcome through use of a nylon or wire guide-stiffener inserted into the tubing prior to the procedure and withdrawn as the tubing is introduced. Impaction of the tubing should be suspected when there is difficulty in advancing tubing which has entered the vein but not reached the mediastinum. This can be confirmed by the ease with which saline can be injected. Repeated impaction indicates need for reinsertion into a more suitable vein. Venous spasm is prohibitive but not likely to appear in the few moments needed to pass the soft catheter into the mediastinum. The use of heparin in the irrigation saline is probably unnecessary in view of the anticoagulant properties of Silastic. Manometric observation indicates whether the tip is located in (1) the right atrium or its tributary veins, (2) the right ventricle, (3) the pulmonary artery and its branches including (4) the pulmonary "wedge" position. The tubing we use contains 10 per cent barium sulfate, permitting fluoroscopic observation although the injection of a small quantity of contrast material may occasionally be desirable.

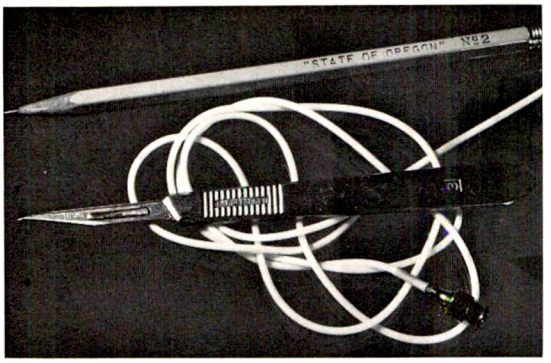


FIG. 1. *Flexible tubing for percutaneous flow-guided cardiac catheterization.* A 40 inch length of Silastic X-30146: i.d. 0.04 inch; o.d. 0.08 inch.

* Work done in connection with this study was performed in the University of Oregon's Minthorn Memorial Laboratory for Cardiovascular Research through Radiology and aided by Grants from the U.S.P.H.S. (H-3275, C3 S1), the Oregon Heart Association and the Mallinckrodt Chemical Works.

Presented at the Sixty-second Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 26–29, 1961.

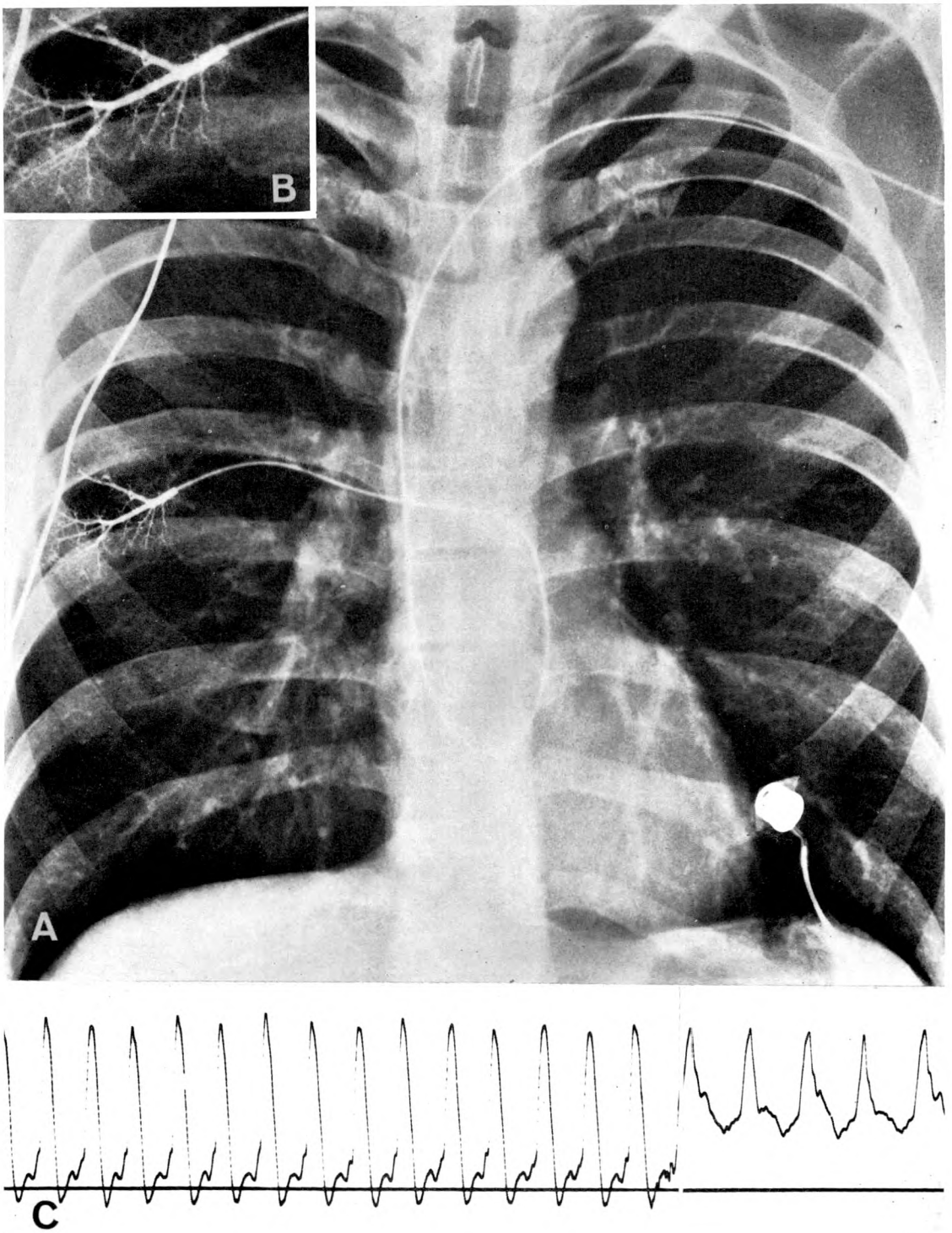


FIG. 2. *Flow guided Silastic catheter used for wedge pulmonary angiography.* Normal 42 year old male. (*A*) Roentgenogram during injection of 3 cc. Conray, catheter having been passed via veins and right heart chambers without use of fluoroscopy. (*B*) The normal wedge angiogram reproduced at actual size. (*C*) Right ventricular and pulmonary arterial pressures recorded during procedure.

DISCUSSION

This catheter may be used for most of the purposes of conventional cardiac catheterization. Figure 1 illustrates the tubing, Figure 2, its roentgenographic appearance during pulmonary wedge angiography, and Figures 3 and 4 representative pressure records. Used approximately 25 times, flow-guided catheterization failed twice in patients whose veins had seriously suffered during protracted prior hospitalization. Occasionally, the right ventricle was not reached due to coiling in the right atrium. Silastic tubing may be autoclaved or boiled and is chemically inert for practical purposes. An ordinary large bore hypodermic needle should not be used for introducing Silastic tubing into veins, since careless or inadvertent motion could result in severance of the tubing and embolization, a complication which has not yet arisen but will likely emerge as a specific hazard if the method receives widespread application. It is also probable that, like other intracardiac cathethers, soft ones will manage to get themselves tied around chordae tendineae and require surgery for removal. To reject the method for such reasons would not seem justifiable.

The possibilities inherent in this technique are considerable. The essential feature of venous (right heart) catheterization is now available for use on a ward or office basis and provides simple means for the

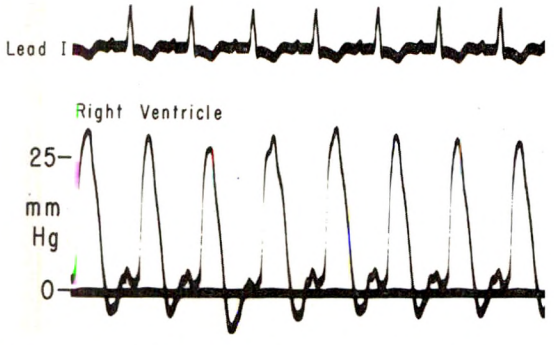


Fig. 3. *Right ventricular pressure record obtained through flow-guided flexible tubing.* Electrocardiogram above; Statham P23 strain gauge and Electronics for Medicine System. Only slight—clinically unimportant—damping is evident.

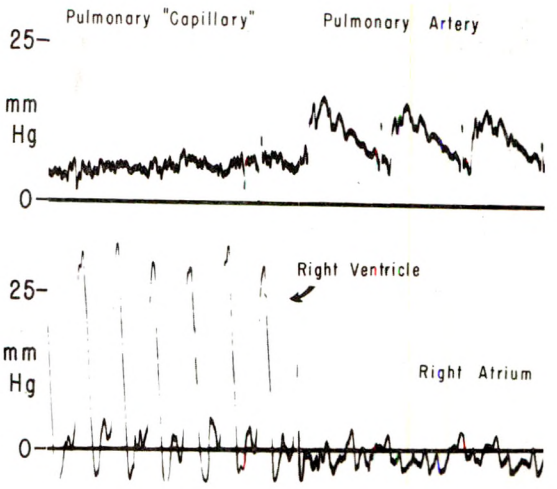


Fig. 4. *Blood pressure records obtained through flow-guided Silastic tubing. Above:* Pulmonary "capillary" and arterial pressures in one patient. *Below:* Continuous "pull-out" pressure record during withdrawal of catheter from right ventricle to right atrium in a patient with scleroderma. The ventricular systolic pressure is "high-normal."

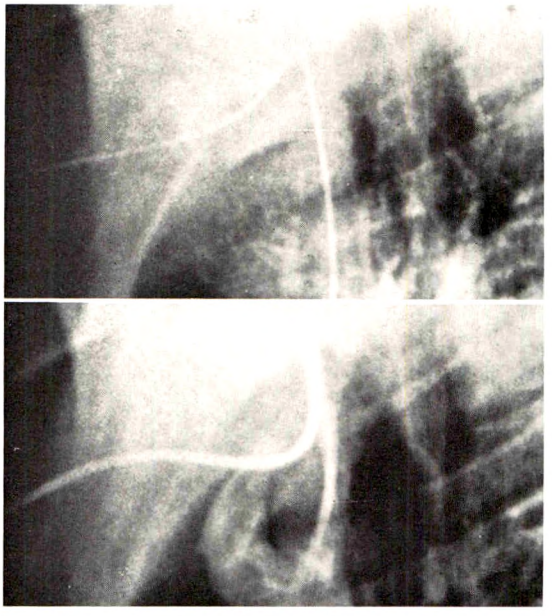


Fig. 5. *Distention of Silastic tubing during injection.* Compare small caliber before injection (*above*) to the distended lumen present during injection (*below*). This promising approach warrants further study to determine optimum values for catheter elasticity, tensile strength, lumen size, and length, related to the viscosity and volume of injected fluid and the force and duration of injection.

selective injection of therapeutic or diagnostic agents including marker dyes. A sterile package of clear, flexible plastic film containing tubing, introductory needle and syringe would permit the measurement of pulmonary arterial pressure as part of the routine studies done on hospital admission. The tubing could then be withdrawn to a suitable extracardiac position and left in place, obviating further trauma due to venapuncture during the hospitalization, simplifying the collection of blood or laboratory study and lowering the incidence of cross-contamination and the costs of hospital operation. Although now feasible, the ward or office determination of pulmonary artery blood pressure will not become a simple matter until suitable transducers and read-out instruments are available. Given the demand, these will be developed.

Within limitations, the flexibility and distensibility of elastic tubing permit rapid injections through relatively small bore tubes (Fig. 5). The extent to which this approach may be applied in angiography remains to be determined. In addition to the present application and those already proved in plastic surgery, neurosurgery, ophthalmology and other fields where permanent compatibility with human tissues is a requirement, Silastic offers major, as yet unrecognized, contributions to medicine. It is suitable for the long term selective administration of chemotherapeutic agents. We are currently exploring its use as an intra-arterial "sleeve" graft introduced and positioned percutaneously and also the use of flow guided catheterization in systemic arteries by means of a short segment of Silastic (.05 inch, o.d.) at the tip of a conventional plastic catheter (a "flip tip" catheter).

CONCLUSION

Flexible silicone rubber tubing has been introduced percutaneously into peripheral veins for the speedy, flow-guided catheterization of systemic veins, right heart and pulmonary vessels. In comparison to conventional right heart catheterization with rigid catheters, the technique may prove to be simpler, safer, less expensive, faster and approximately equal in usefulness. It does not require fluoroscopy.

In addition to the uses to which ordinary cardiac catheters are put, the chemical inertness and elasticity of Silastic open up a number of new and attractive possibilities for diagnosis and treatment.

Contingent upon the development of suitable packaging techniques and appropriate read-out instruments, the routine determination of pulmonary artery pressure can become a practical reality.

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The generosity and knowledge of Dr. R. R. McGregor, Mr. S. A. Braley and Dr. Ethel Mullison of the Dow Corning Center for Aid to Medical Research are gratefully acknowledged.

CO_2 ANGIOCARDIOGRAPHY*

IMPROVED TECHNIQUE AND RESULTS

By MANUEL VIAMONTE, JR., M.D.†
MIAMI, FLORIDA

CO_2 angiocardiography is the roentgenographic study of the heart with the use of pure carbon dioxide as a negative contrast medium. This technique was introduced by Paul *et al.*[10] Since 1957 very few articles have been published in reference to its clinical use.[1,3,11,14] It is the purpose of this paper to present our technique and to illustrate some of its indications.

TECHNIQUE

Carbon dioxide is 20 times more soluble in plasma at body temperature than air or oxygen.[12] It will persist as a gas long enough to permit roentgenographic visualization. Experiments on dogs have proven that large amounts of CO_2 (as much as 15 l.) can be injected into the blood stream within a 5 minute period, with relatively few side effects.[17] These rapid injections cause a brief rise in whole blood CO_2 content of 5–10 volumes per cent, and a 1–2 per cent decrease of oxygen concentration, with an associated increase in the depth and rate of respiration. Large injections of CO_2 produce only minimal changes of the intravascular pressures. Prolonged blood clotting time has also been reported.[4]

We have injected mongrel dogs using different positions and with variable amounts of CO_2, sometimes far beyond the amount needed for adequate intracardiac visualization in humans. Our injections have included the left side of the heart and the aorta. After injections in the aorta, S-T and T changes have transiently and invariably occurred. No deaths have been noted in our animal experiments (more than 100 injections), and none after 50 injections in humans. Only one death has been reported in the literature.[7] It is questionable if this can be attributed directly to the CO_2 injected.

Pure carbon dioxide injected into an antecubital vein of a patient lying in the left lateral decubitus position will enter the right atrium through the superior vena cava and will be temporarily trapped in the right atrium[8] (Fig. 1). When the right atrium contains CO_2, its lateral wall appears in the frontal projection as a smooth, uniformly thick, convex, opaque band, not greater than 4 mm. in thickness. The radiopaque band separates the radiolucency produced by the CO_2 inside the right atrium from the radiolucency adjacent to the right heart border and determined by the air in the right lung. This opaque band is a composite shadow formed by the superimposition of the endocardium, myocardium, the two layers of the pericardium and the mediastinal pleura (Fig. 2). Before doing CO_2 angiocardiography for the evaluation of pericardial pathology, it must be determined that a pleural effusion is not present on the right side. If right pleural fluid is found, this fluid will overlie the heart when the patient lies on the left side, and an erroneous impression of an abnormally thickened right cardiac border may be gotten. In this case the pleural effusion and not pericardial pathology will be the cause of the abnormality.

Preliminary roentgenograms before a CO_2 angiocardiographic study should include posteroanterior and lateral roentgenograms of the chest, and if necessary a posteroanterior view in the right lateral decubitus with horizontal beam in order to rule out the presence of right pleural effusion. The beam is centered to the midline at a point equidistant from the suprasternal

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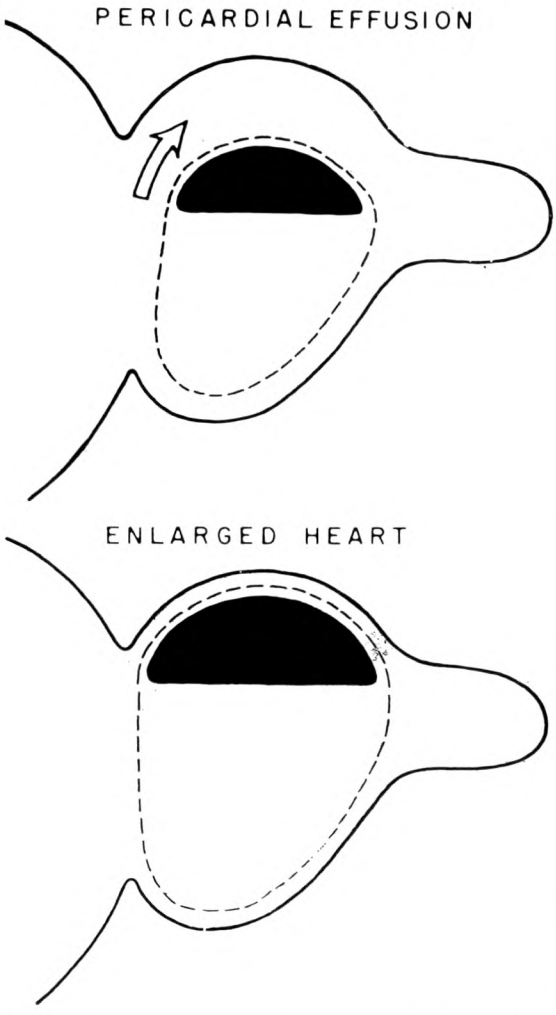


Fig. 1. Diagrams of two enlarged heart shadows. *Upper:* Pericardial effusion showing thickened right heart border. *Lower:* Cardiac dilatation without effusion showing normal right heart border. (Reproduced by courtesy of *Radiology*.[11])

notch to the xiphoid process. The following equipment is necessary for the intravascular injection of CO_2 (Fig. 3, 4 and 5).

1. A 100 cc. BD syringe with a central metallic tip and a Luer-Lok end.
2. A rubber tubing with two adaptors, one on each end (Fig. 4A).
3. A 16 or 18 gauge needle, $\frac{1}{2}$ to $\frac{3}{4}$ of an inch long.
4. A tank containing 100 per cent pure CO_2 with a meter and a long tube (Fig. 4B).
5. Isotonic saline solution and intravenous tubing (Fig. 4C).
6. A 10 cc. syringe.
7. Alcohol sponges.
8. A tourniquet.
9. A special set of stopcocks, which is illustrated in Figure 3.

After obtaining and studying the preliminary roentgenograms, the patient is placed in the left lateral decubitus position with 10° to 15° Trendelenburg tilting (Fig. 5). A needle is inserted into an antecubital vein of the dependent left arm. The needle and vein are kept patent by the slow injection of isotonic saline solution. The 100 cc. syringe with tubing attached is flushed several times with pure 100 per cent CO_2 in order to eliminate all the air in the system (Fig 6, *A*, *B* and *C*). The syringe is filled with 100 cc. of CO_2 and kept under continuous hand pressure after closing the stopcock located at the end of the tubing (Fig. 6*D*). When this has been accomplished the mainswitch of the roentgenographic machine is turned on and the patient is di-

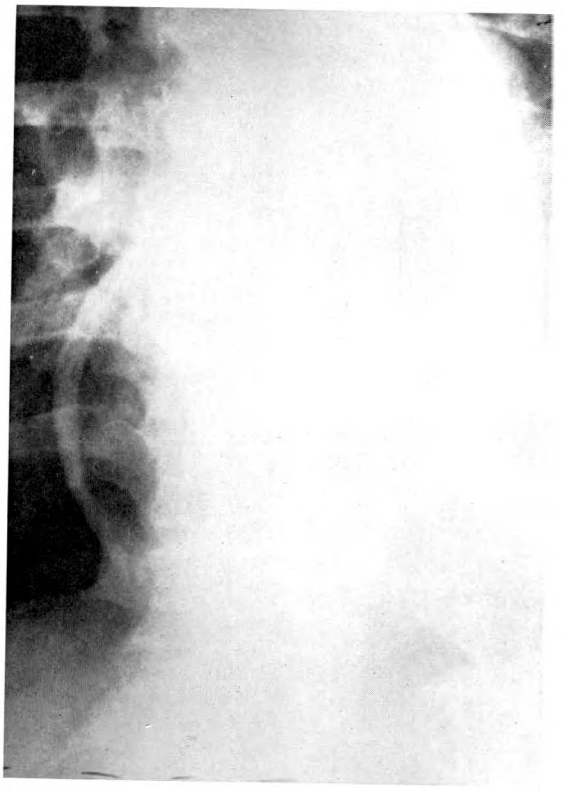


Fig. 2. A normal CO_2 angiocardiogram.

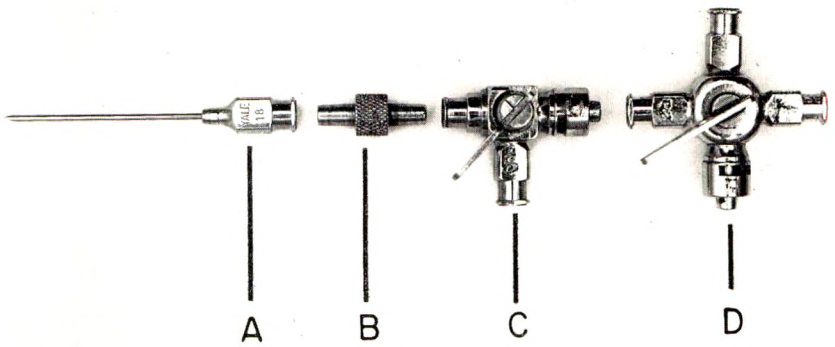


Fig. 3. Special set for CO_2 angiocardiography. A: 18 gauge needle; B: adapter—male Luer-Lok to male Luer-Lok; C: three-way stopcock—female Luer-Lok to male Luer-Lok to female Luer-Lok side connection with large bore; D: four-way stop cock—three female Luer-Loks, one male Luer-Lok.

rected to inspire deeply, and to hold the breath without straining. The stopcock is then opened and within 2–3 seconds a total of 100 cc. is injected (Fig. 6*E*). At the completion of the injection, the roentgenogram is taken. After this, the intravenous injection can be continued until the viewing of the roentgenograms. The patient is kept in the same position for the following 5 or 10 minutes. A second and third injection may be made if necessary.

CLINICAL APPLICATION

Intravenous carbon dioxide injection can be used safely and conveniently for demonstrating free pericardial effusion (Fig. 7), fibrous pericarditis (Fig. 8) and pericardial cysts (Fig. 9, *A* and *B*) over the right heart border. Pericardial effusion will produce a uniformly, round, thickened band (more than 4 mm.). Fibrous pericarditis involving the right heart border can be determined by an irregularly thickened and flattened opaque band with or without associated pericardial calcification.

CO_2 angiocardiography is of particular value in patients when a simple and rapid investigation of the pericardium is required. Expensive and complex angiocardiographic equipment is not necessary.[16] When a more thorough study is recommended, particularly for underlying heart disease, opaque angiocardiography should be the procedure of choice.

CO_2 angiocardiography assists in the evaluation of the superior vena cava and juxtacardiac portion of the inferior vena

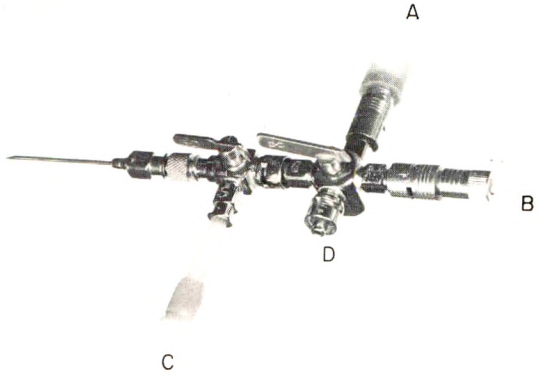


Fig. 4. Interconnections of special set for CO_2 angiocardiography. A: Tubing leading to 100 cc. large tip bore Luer-Lok Robb-Steinberg syringe; B: tubing to CO_2 tank; C: tubing to intravenous solution container; D: side outlet for air removal.

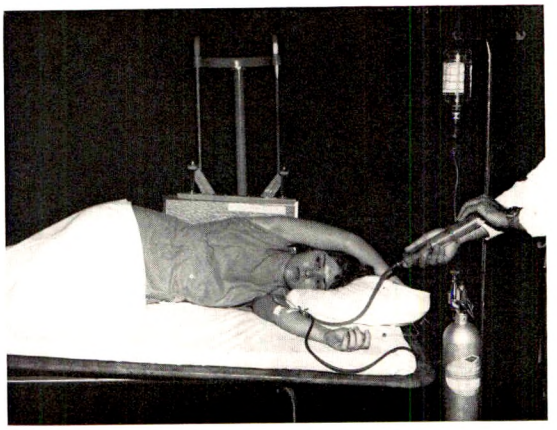


Fig. 5. Positioning of the patient for single roentgenographic CO_2 angiocardiography.

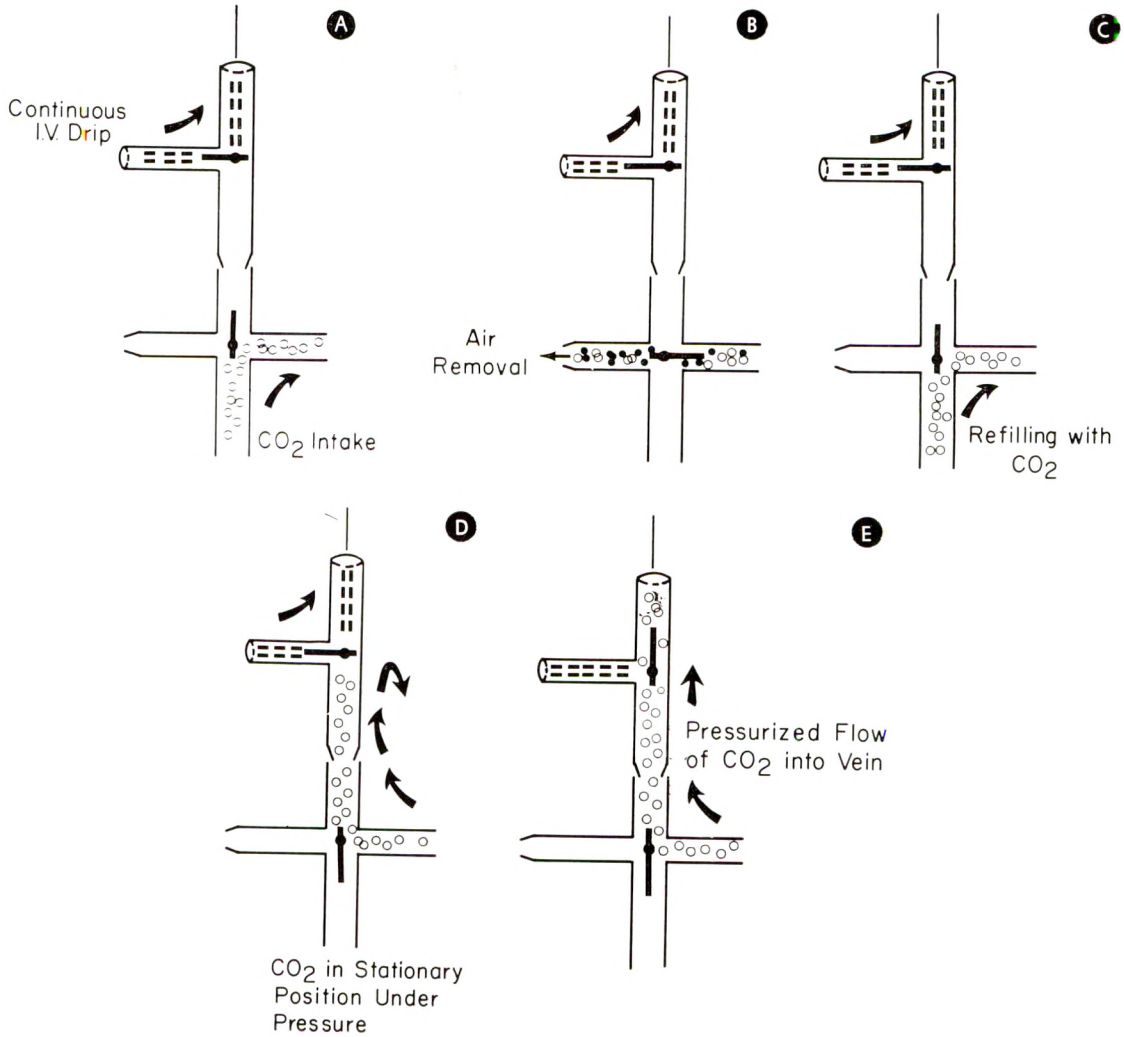


Fig. 6. Diagrams illustrating the different steps of injection. (*A*) CO_2 intake into the syringe. (*B*) Air removal through side outlet. (*C*) Refilling with CO_2. (*D*) CO_2 in stationary position and under pressure. (*E*) Injection of CO_2 into vein after discontinuing the intravenous drip.

cava.[5] We recently had a false positive case of abnormally thick right heart border that we thought was related to pericardial effusion. Postmortem examination showed no pericardial effusion and a very thick pad of fat surrounding the heart.

Experimentally, we have used CO_2 angiocardiography in the study of the atrioventricular valves and also in the evaluation of the internal architecture of the 4 chambers of the heart (Fig. 10). We have also injected the ascending aorta for the demonstration of the coronary arteries. Intracardiac shunts, especially at the atrial level, have been evaluated experimentally.[17,18]

A contraindication to CO_2 angiocardiography is the presence of severe pulmonary emphysema with fibrosis.[2] These patients will have difficulty in metabolizing the CO_2 that is injected due to the advanced and severe chronic lung disease. Poor general condition of the patient and inability to cooperate are relative contraindications of the procedure.

The only significant complication of CO_2

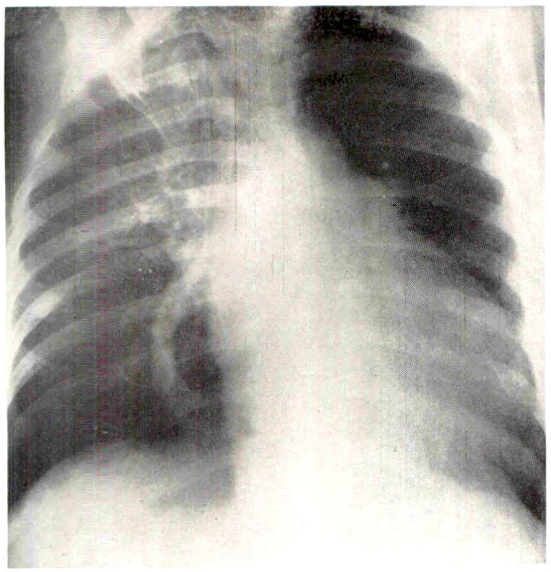


FIG. 7. CO_2 angiocardiogram showing effects of pericardial effusion. The opaque band measures 9 mm.

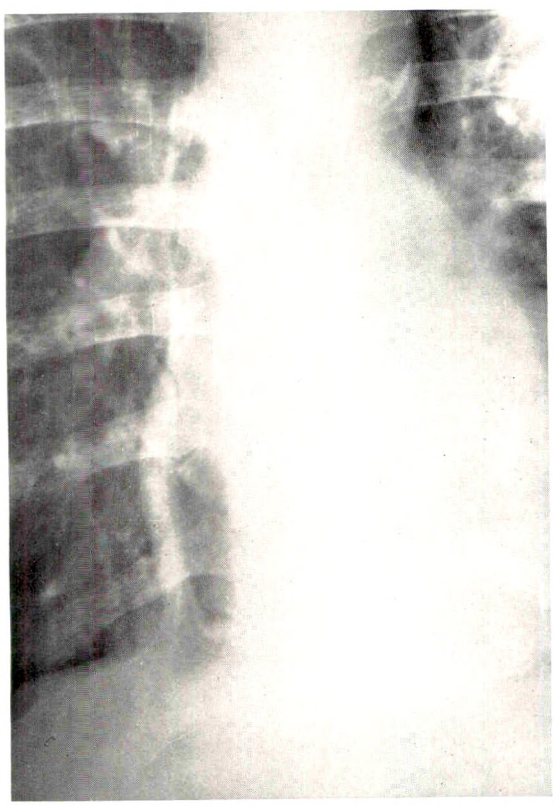
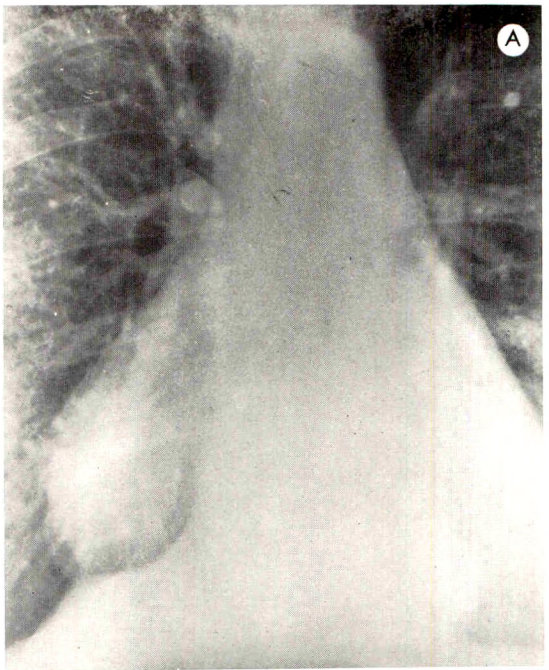


FIG. 8. CO_2 angiocardiogram showing findings compatible with fibrous pericarditis. Note flattening and thickening of the opaque band.



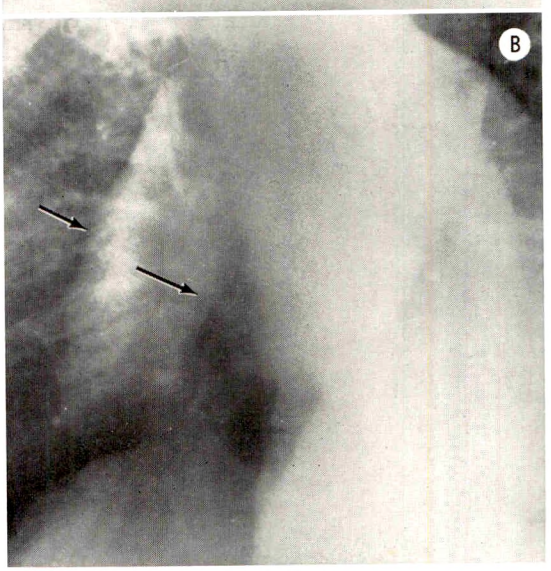


FIG. 9. Pericardial cyst. (*A*) Abnormal density adjacent to right heart border. (*B*) CO_2 angiocardiogram showing extracardiac location of the mass.

angiocardiography is the injection of air and the subsequent production of embolism which may be fatal. By using a proper technique, this serious risk can be prevented.

Most of the published papers about CO_2 angiocardiography are based on experi-

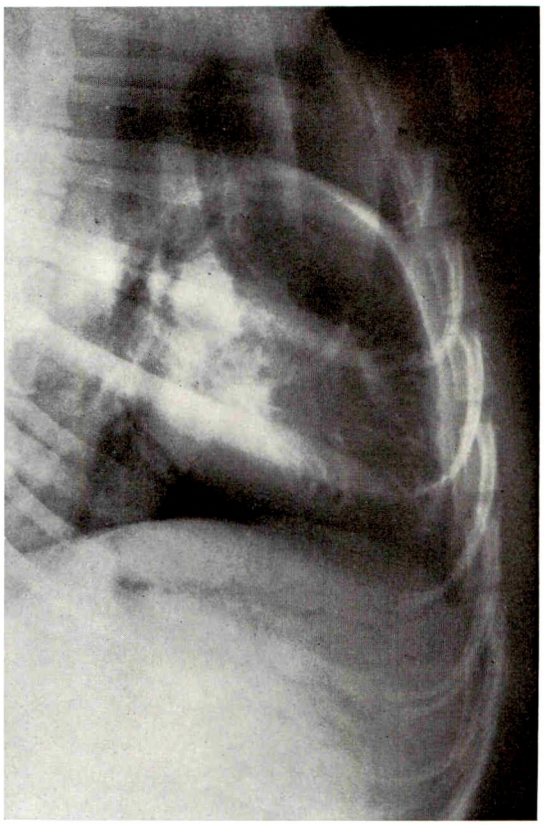


Fig. 10. CO_2 angiocardiogram in a dog made in the supine position. Note the atrioventricular valves and detail of the internal architecture of the right ventricle.

mental work with dogs. All kinds of anatomic and physiologic studies have been made using various amounts of CO_2 and obtaining roentgenograms in different positions. Cinefluorographic examinations have been carried out and even the coronary arteries have been injected with CO_2.[9,13] Recently, Martin, Meredith and Johnston[6] published an interesting experimental study of the heart.[6] They injected simultaneously CO_2 and an opaque medium in order to obtain a double contrast study of the intracardiac structures and feel that this technique has advantages from which a clinical application may be derived. We have also tried simultaneous and successive injections of gas and opaque contrast material,[6] and so far have been unsuccessful. We believe that small amounts of opaque contrast material or pure CO_2 injected alone will give enough detail of the intracardiac chambers and that double contrast angiocardiography does not have the same merit as the independent injections of opaque or of negative contrast material.

For our selective injections of CO_2, we have used the intercalative apparatus of Dr. Boucek and Dr. Murphy, which permits a rapid injection and also a synchronization of the trigger mechanism of the roentgenographic unit with the electrocardiogram, which is utilized as an activating impulse. Again, the safety of the intravascular CO_2 injections has been demonstrated through these experiments.

The main indications for CO_2 angiocardiography are:

1. Detection of pericardial effusion.
2. Demonstration of fibrous pericarditis at the right atrial level.
3. Evaluation of paracardiac masses adjacent to the right atrium.
4. Study of right atrial pathology.
5. Examination of superior vena cava and juxtacardiac portion of the inferior vena cava.

CONCLUSION

CO_2 angiocardiography should become a more accepted diagnostic procedure. It is safe and simple to perform. Its main indication will be in the evaluation of pericardial and right heart pathology.

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BILATERAL SIMULTANEOUS INTRAVENOUS ANGIOCARDIOGRAPHY*

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THE method of rapid, bilateral, and simultaneous intravenous injection of concentrated organic contrast media was primarily devised for abdominal arteriography, peripheral arteriography, and cerebral angiography.[14,22] In a 2 year period, during which 500 patients have had bilateral simultaneous intravenous injections, 87 cases had bilateral intravenous injections for assessment of cardiovascular disease. Analysis of the findings in these cases form the basis of this report and provide data for determination when the double, simultaneous, intravenous technique is indicated for cardiovascular angiography.

METHOD

The Robb-Steinberg method for intravenous angiocardiography by rapid injection of concentrated organic iodides and precisely timed roentgenography is the foundation of the study herein reported.[5,7,8,11,13] Modification of the method by the simultaneous rapid injection of contrast media into both arms via special 12 gauge Robb-Steinberg cannulas (total dose 1 ml. per kg. of body weight divided equally into the two special 12 gauge syringes) permitted the contrast agent to act as undiluted bolus and opacification of the entire circulatory system was achieved.[14,16–18,20–24] Angiocardiography, cerebral angiography, abdominal aortography, and peripheral arteriography followed; roentgenographic visualization of each part was accomplished depending upon the time these structures were opacified.

Prediction of the time of opacification of the various components of the cardiovascular system was determined by a preliminary modified circulation time made by injecting diluted sodium dehydrochlorate via one of the special needle stop-cock units.[14,22] The bitter taste accurately foretold the opacification time of the arch of the aorta. For angiocardiography, the patient was usually examined in the seated, erect frontal and left anterior oblique positions. This necessitated two series of double injections of contrast medium. All were well tolerated even though regularly repeated at short intervals. In some of the cases, simultaneous biplane studies in the supine position (frontal and lateral views) were made; in such instances only the single series of double injections sufficed. Serial angiocardiograms (14 by 14 inch cut film) were secured with the Schönander apparatus The time intervals varied from 2 to 6 films per second in congenital heart disease to intervals of a film every 0.75 second or longer in acquired heart disease, for the duration of the circulation time. No outward reaction occurred in this group.

RESULTS

Twenty-three (26 per cent) of the 87 patients in whom bilateral simultaneous intravenous angiocardiography was performed had buckling of the brachiocephalic arteries.[22] Fourteen (16 per cent) of the cases were studied to delineate large thoracic aortic aneurysms (Fig. 1, *A* and *B*; and 2, *A–D*), while 10 patients (8.7 per cent) had huge hearts owing to congestive heart failure (Fig. 3, *A* and *B*). Nine patients with acquired and congenital aortic valvular lesions were studied (Fig. 4, *A–D*; and 5, *A*, *B* and *C*). Coarctation of the aorta, especially in heavy individuals, was the indication for bilateral injection in 7 instances. Peripheral vascular complica-

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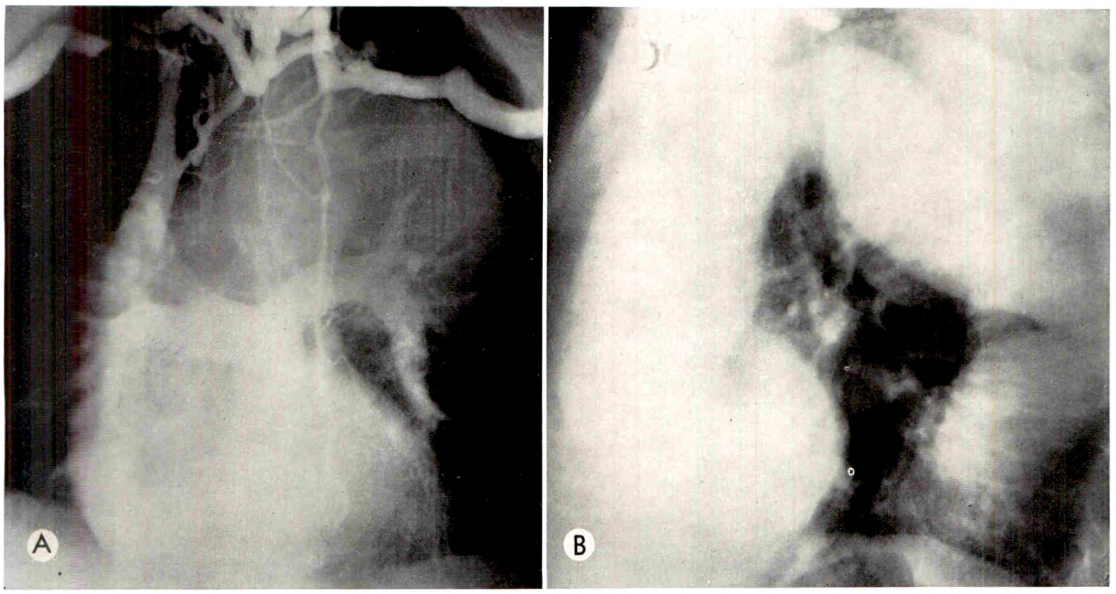


Fig. 1. Large (190 lb., 6 ft.) 61 year old man with huge syphilitic aneurysm of the thoracic aorta. (*A*) Frontal angiocardiogram showing occlusion of the left innominate vein and collaterals about the neck. (*B*) Left anterior oblique angiocardiogram showing the dilated ascending aorta and the saccular aneurysm of the arch and descending aorta.

tions were demonstrated in 5 patients with rheumatic heart disease, usually mitral stenosis (Fig. 6, *A* and *B*; 7, *A* and *B*; and 8, *A* and *B*). Three patients with large goiters, 2 patients with massive pericardial effusions, 2 individuals with huge pericardial effusions and large hearts, and 7 persons with congenital heart disease (Fig. 9, *A* and *B*) constituted other groups. Finally, a miscellaneous collection of 7 patients had varied conditions such as anomalous arteries from the aorta to the lungs (4 cases), thoracic deformities (2 cases), and an unusual instance of a congenital arteriovenous fistula of the right thyrocervical vessels (Fig. 10, *A* and *B*).

DISCUSSION

Figure 1, *A* and *B* shows an overweight patient with a huge syphilitic aneurysm of the thoracic aorta. The left innominate vein was occluded by the aneurysm (Fig. 1*A*). The dilated thoracic aorta and the aneurysm of the arch are evident (Fig. 1*B*). Another huge (congenital) aneurysm arising from the under surface of the transverse portion of the thoracic aorta is shown in Figure 2, *A–D*. Originally this case was reported in 1951.[3] The present angiocardiographic studies made in 1960 followed increasing dyspnea and enlargement of the aneurysm. The diagnosis was confirmed by postmortem examination. The marked pulmonary arterial deformities (Fig. 2, *A* and *C*) are striking.

In a patient with a huge dilated heart and great blood vessels owing to hypertension and chronic heart failure, the markedly dilated left heart chambers and ascending aorta were clearly visualized (Fig. 3, *A* and *B*). A patient with the skeletal and ocular stigmata of Marfan's syndrome, who had been mistakenly diagnosed as having aortic regurgitation owing to rheumatic fever, is shown in Figure 4, *A–D*. The double-barreled intravenous angiocardiographic studies disclosed a hugely dilated ascending aortic aneurysm involving the aortic sinuses. The aneurysm produced a characteristic filling defect of the superior vena cava, right atrium, and outflow tracts of the right ventricle (Fig. 4, *B* and *D*).[19] Retrograde filling of the azygos vein was apparently related to pressure exerted on the

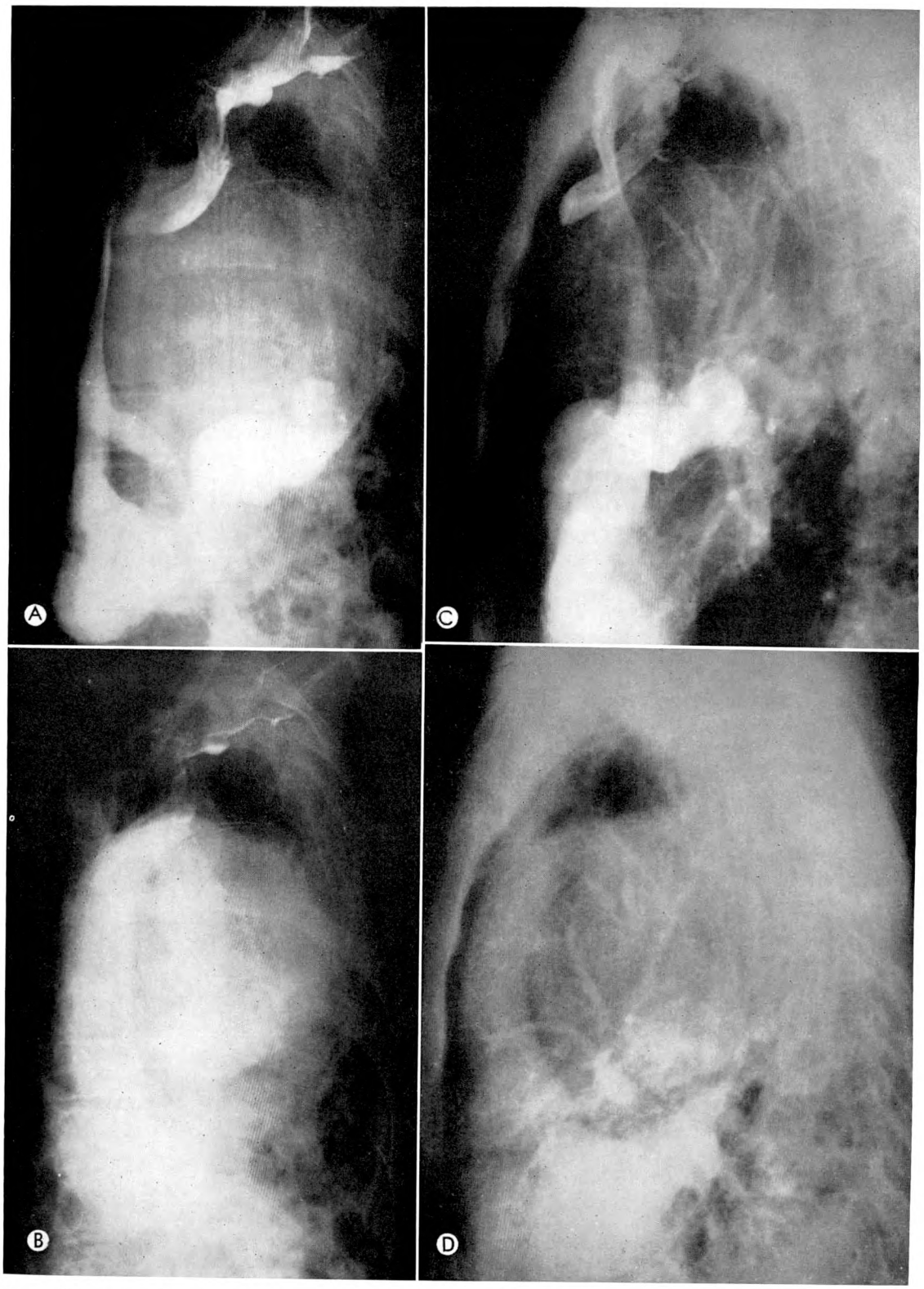


FIG. 2. Huge saccular, congenital aneurysm of the undersurface of the aortic arch (confirmed by autopsy) in a 38 year old man. (*A*) Frontal angiocardiogram showing deformity of the pulmonary artery and branches. (*B*) Frontal angiocardiogram showing the intact ascending aorta and opacified saccular aneurysm of the transverse portion of the aorta. (*C*) Lateral angiocardiogram revealing the downwardly displaced pulmonary artery and branches. (*D*) Lateral view of the normal ascending aorta demonstrating that the aneurysm arises from the undersurface of the aortic arch.

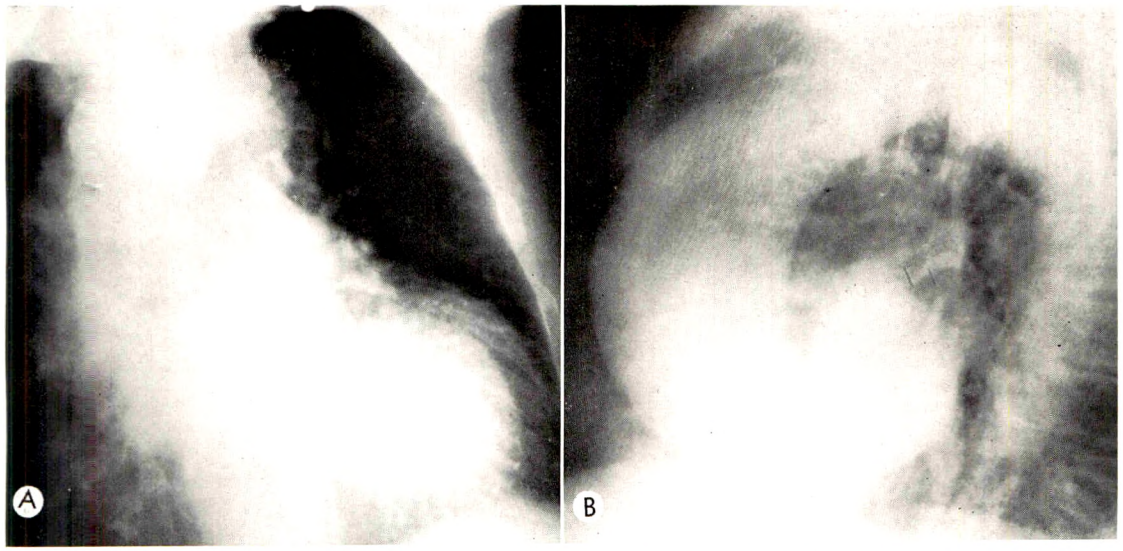


Fig. 3. Cardiomegaly in a short, heavy, 38 year old man with hypertension, chronic heart failure, and aneurysmal dilatation of ascending aorta. (*A*) Frontal angiocardiogram showing enlargement of the left atrium, ventricle (wall and cavity) and the dilated ascending aorta. (*B*) The left anterior oblique angiocardiogram of the left heart and aorta.

superior venae cava. The hugely dilated left ventricular cavity was a reflection of aortic valvular insufficiency. Another patient with a huge aneurysm of the ascending aorta involving the aortic sinuses and also producing aortic insufficiency is illustrated in Figure 5, *A*, *B* and C. In this instance, none of the clinical manifestations of Marfan's syndrome was present; at autopsy medial cystic degeneration of the aorta was found.

In a patient with peripheral vascular disease (occlusion of the right superficial femoral artery) associated with rheumatic heart disease, the bilateral intravenous insertion of the cannulas permitted excellent visualization of the cardiovascular system and a diagnosis of mitral stenosis (Fig. 6, *A* and *B*). A patient with an enlarged rheumatic heart and peripheral vascular disease (occlusion of the left tibial artery) was shown to have an enlarged right atrium with the "jet sign" of tricuspid regurgitation (Fig. 7, *A* and *B*).[4] Finally, bilateral superior venae cavae were visualized in a woman with rheumatic mitral stenosis (Fig. 8, *A* and *B*).

The 2 patients with congenital heart disease (Fig. 9, *A* and *B*) demonstrate the value of the double injection technique in a child with bilateral superior venae cavae, dextrocardia, and tetralogy of Fallot (Fig. 9*A*) and in a huge overweight adult with a right posterior aortic arch and aortic diverticulum that simulated a mediastinal tumor (Fig. 9*B*). The venous and arterial components of an arteriovenous fistula over the right shoulder were also visualized (Fig. 10, *A* and *B*).

The 7 patients in this series with coarctation of the aorta had the double-barreled injections because, as often occurs in this disease, they were stockily-built and overweight. This does not mean that bilateral intravenous injections are indicated in every patient with coarctation of the aorta. Indeed, Figure 11, *A* and *B* shows how well the aorta can be demonstrated with a single injection in this disease. Further proof of the efficacy of the single intravenous hand injection technique is the fact that, during the 2 year period that the 87 cases in this series of double injection angiocardiography were collected, several hundred patients in this center had angiocardiography with the regular single injection method.

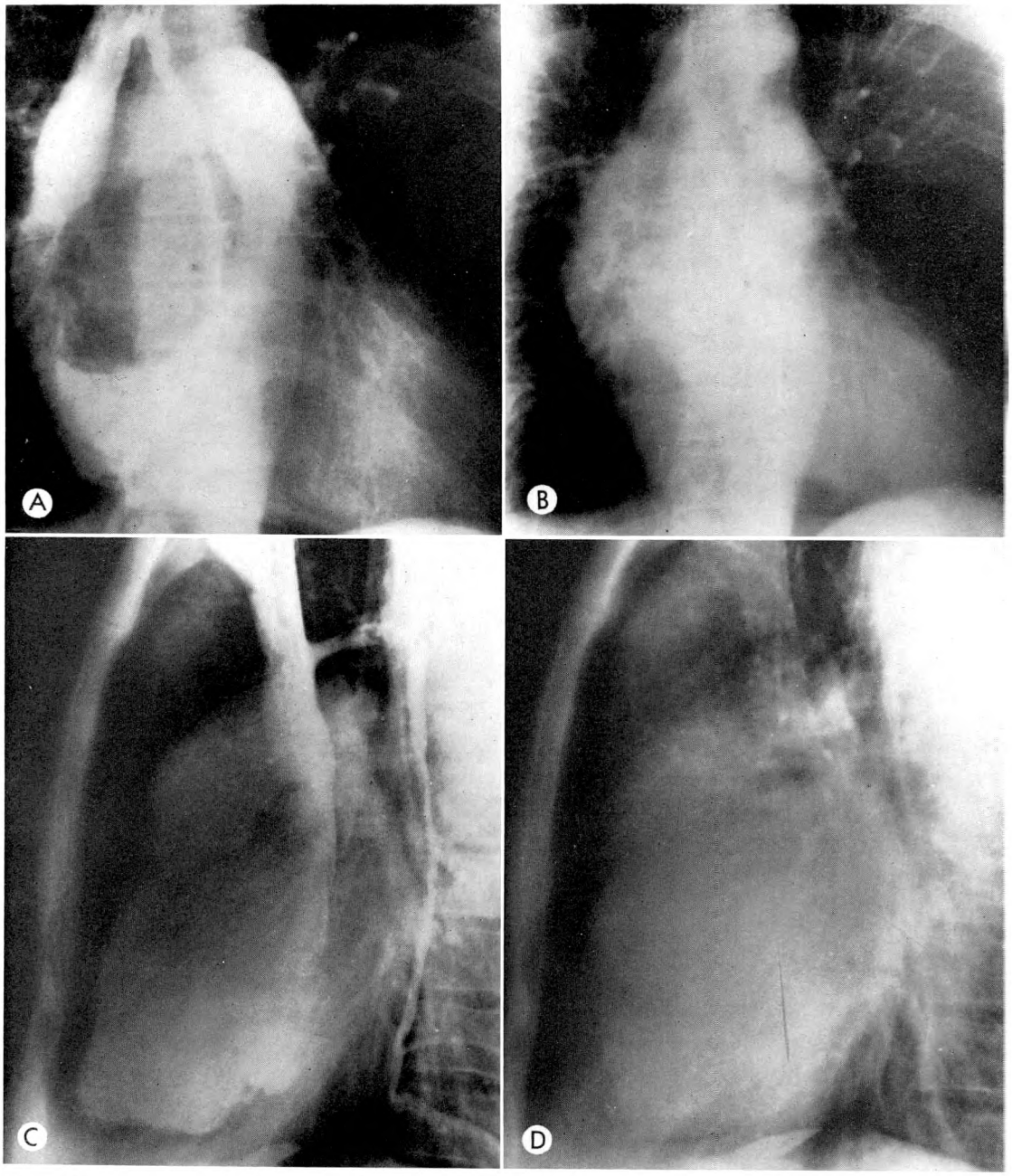


Fig. 4. Biplane angiocardiographic study of a well-developed 42 year old man with classic skeletal and eye manifestations of Marfan's syndrome and aortic regurgitation with chronic heart failure. (*A*) Frontal angiocardiogram showing pressure deformities of the lower end of the superior vena cava, the upper portion of the right atrium, and outflow tract of the right ventricle. There is also reflux filling of the azygos vein. (*B*) Frontal angiocardiogram showing marked aneurysmal dilatation of the ascending aorta involving the aortic sinuses. The left ventricular cavity is unusually dilated owing to aortic valvular regurgitation. (*C*) Biplane lateral angiocardiogram also demonstrating filling defects of the superior vena cava, inflow portion of the right atrium, and outflow tract of the right ventricle. (*D*) Biplane lateral angiocardiogram again demonstrating the markedly dilated ascending aorta, aneurysmal dilatation of the aortic sinus, and dilated left ventricle.

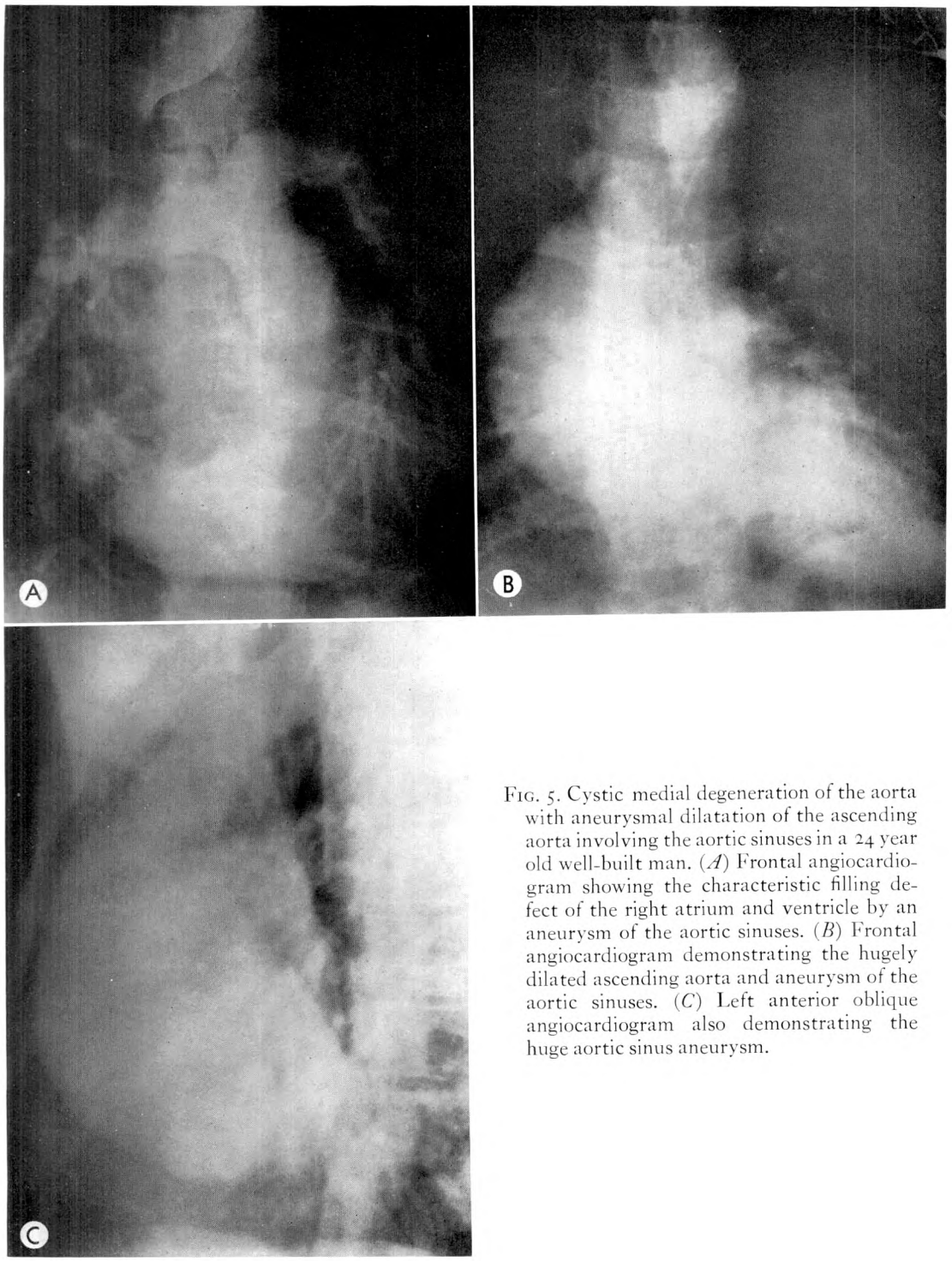


FIG. 5. Cystic medial degeneration of the aorta with aneurysmal dilatation of the ascending aorta involving the aortic sinuses in a 24 year old well-built man. (*A*) Frontal angiocardiogram showing the characteristic filling defect of the right atrium and ventricle by an aneurysm of the aortic sinuses. (*B*) Frontal angiocardiogram demonstrating the hugely dilated ascending aorta and aneurysm of the aortic sinuses. (*C*) Left anterior oblique angiocardiogram also demonstrating the huge aortic sinus aneurysm.

Two deaths (both in elderly men with advanced abdominal aortic and peripheral vascular arteriosclerosis) have occurred in the first 500 patients studied with the method of bilateral simultaneous intravenous injections of concentrated contrast

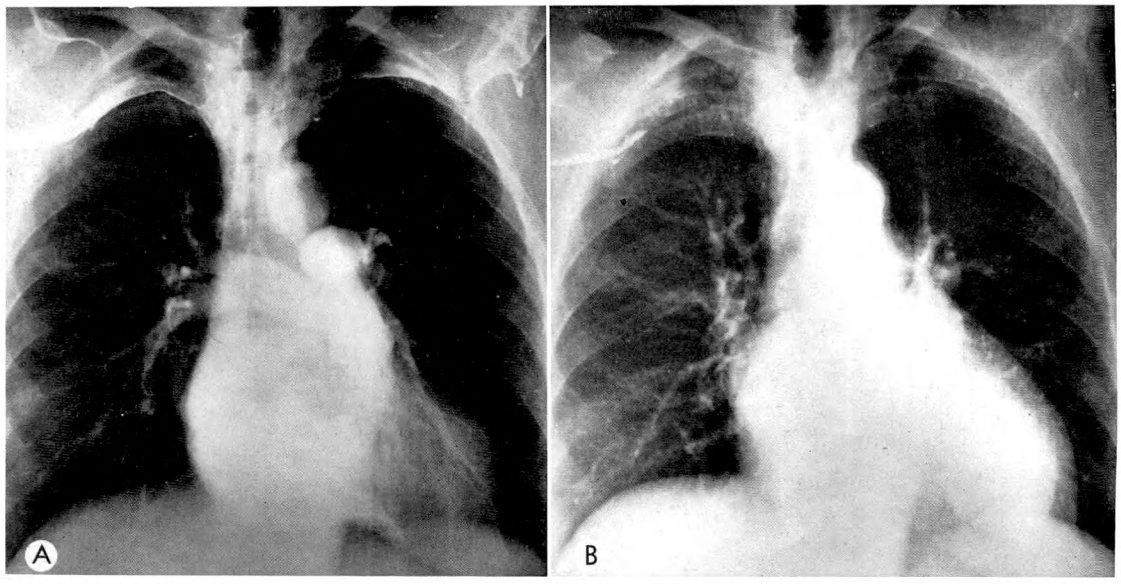


FIG. 6. Classic mitral stenosis of rheumatic fever origin in a woman with peripheral vascular (right superficial femoral arterial occlusion) disease. (*A*) Frontal angiocardiogram showing enlargement of the right ventricle and pulmonary artery. (*B*) Frontal angiocardiogram revealing left atrial enlargement (without thrombi) and a normal-sized left ventricle (chamber and wall).

organic iodides. In the first instance, pulmonary edema occurred several hours following the 2 series of injections.[20,21] In the other, pulmonary edema and cardiac arrest developed soon after the first injections; open heart massage was unsuccessful in reviving the patient.[24] Autopsy examination showed pulmonary edema in both cases but

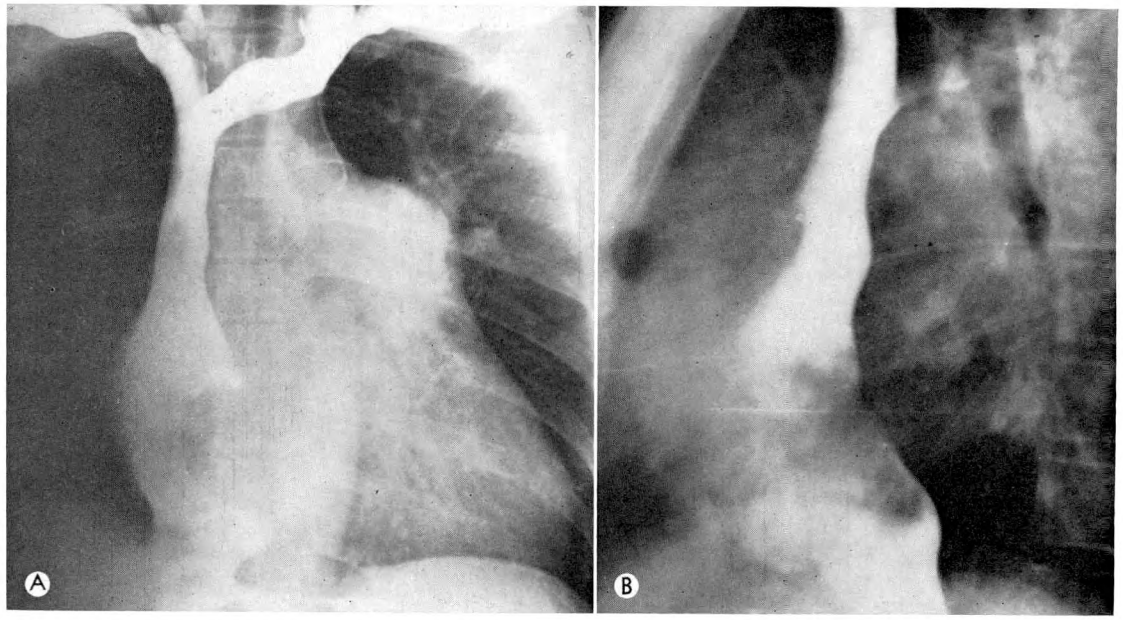


FIG. 7. Demonstration of tricuspid regurgitation in a short obese 51 year old woman with rheumatic mitral stenosis, atrial fibrillation, and peripheral vascular disease. (*A*) Frontal angiocardiogram showing the characteristically enlarged right atrium with a filling defect opposite the tricuspid valve owing to tricuspid regurgitation. (*B*) Biplane lateral angiocardiogram demonstrating the "jet sign" of tricuspid regurgitation.

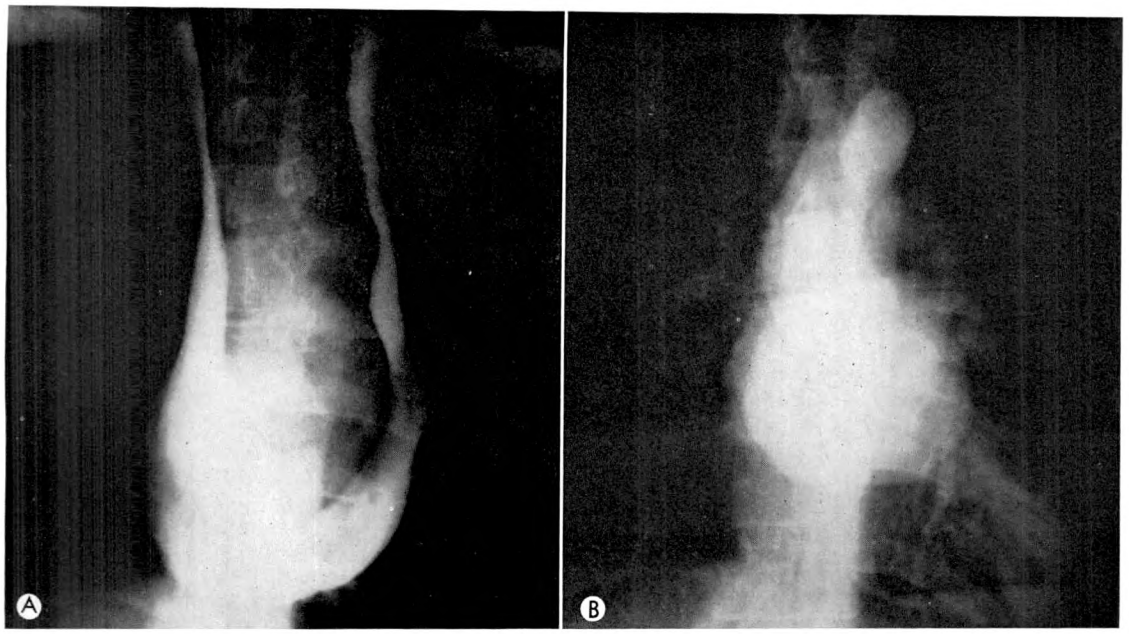


Fig. 8. Bilateral superior venae cavae in a 33 year old woman with rheumatic mitral stenosis. (*A*) Frontal angiocardiogram showing the classic course of a persistent left superior vena cava into the coronary sinus. (*B*) The enlarged left atrium and normal-sized left ventricle of mitral stenosis.

failed to reveal the cause of death. Read,[6] on the basis of animal experiments, has concluded that rapidly injected concentrated organic iodide solutions were capable of producing pulmonary hypertension and fatal acute cor pulmonale. Since death has been reported in 3 instances when only 1 ml. of a contrast agent was used as a test dose,[5] the theory that individuals succumb because of drug sensitivity appears to be more accurate. Because only 2 deaths have occurred in several thousand cases of angiocardiography performed with a single intravenous injection,[12,15] it may be that angio-

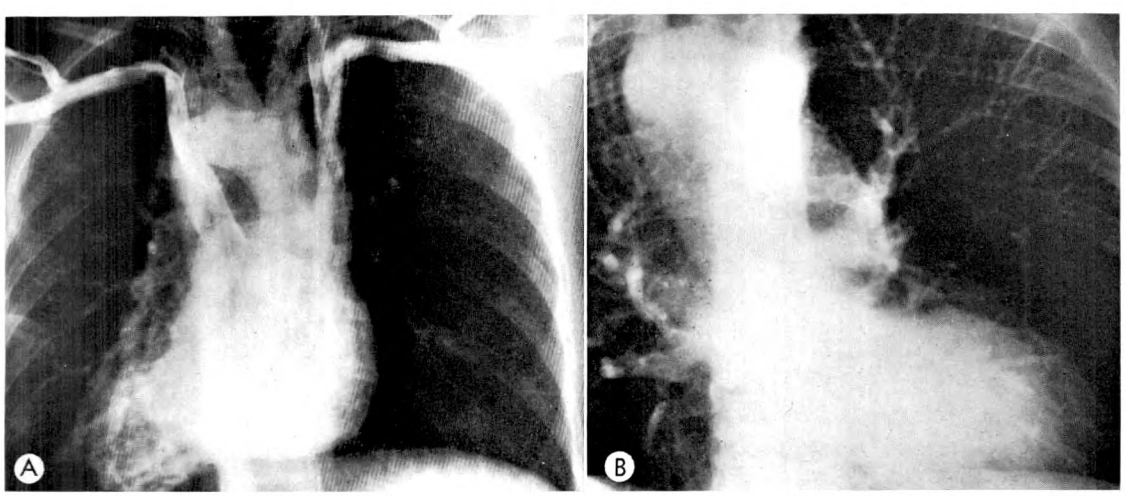


Fig. 9. Value of bilateral, simultaneous, intravenous angiocardiography in congenital heart disease. (*A*) Frontal angiocardiogram in a 10 year old girl showing bilateral superior venae cavae, dextrocardia, and tetralogy of Fallot. (*B*) Angiocardiogram in a 58 year old man demonstrating the right posterior aortic arch with diverticulum simulating a mediastinal tumor.

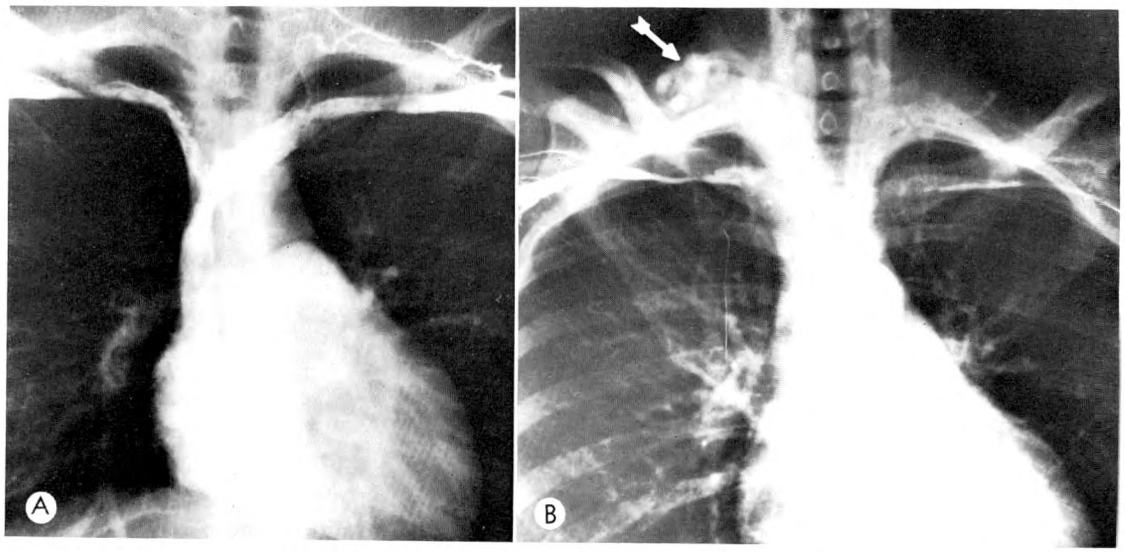


Fig. 10. Arteriovenous fistula of the right thyrocervical vessels. (*A*) Frontal angiocardiogram showing narrowing and mixing defects of the right subclavian and innominate veins. (*B*) When the cardiovascular structures of the left heart are opacified, there is a large cluster of vessels (arteries and veins) to the right of the neck (arrow).

cardiography with the simultaneous bilateral intravenous injection technique is probably more hazardous.

Recently, several papers reported a modification of the double intravenous injection technique of angiocardiography, abdominal aortography, peripheral arteriography and carotid-vertebral arteriography.[1,2,9,10,25] Since these methods require catheter insertions into the arm veins,

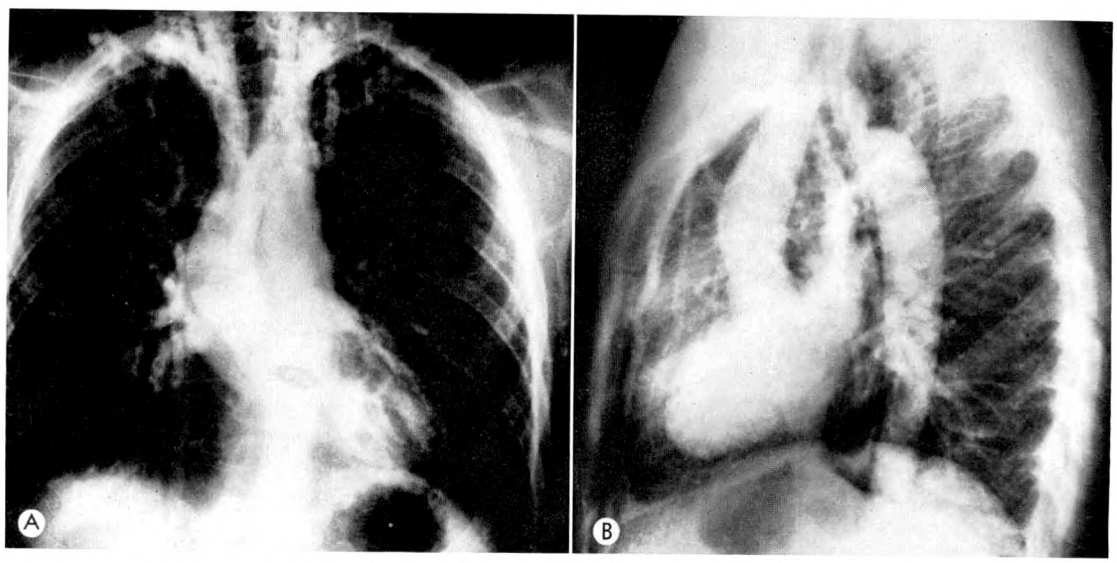


Fig. 11. Angiocardiographic demonstration of coarctation of the aorta with a single intravenous injection in a 12 year old boy. (*A*) Frontal angiocardiogram showing dilatation of the ascending aorta, coarctation of the aorta just beyond the origin of the left subclavian artery, and a plethora of collateral arteries in the neck, axilla and sternal areas. (*B*) Left anterior oblique angiocardiogram also revealing all the classic details of coarctation of the aorta.

vena cava, or right atrium, and often a mechanical injector, they are not as readily available for use in the average small hospital. It would appear, therefore, that the double-barreled intravenous injection technique described above is a more practical procedure.

The advantages of the bilateral simultaneous, intravenous hand injections of concentrated contrast agents for angiocardiography over the single intravenous injection are as follows: (1) for obese and heavy individuals increased opacification results with the double injections because doses of contrast material up to 100 cc. can be given, whereas with the single intravenous hand injection these are limited to 50 cc.; (2) in patients with markedly dilated or hypertrophied cardiac chambers or great vessels (owing to heart failure, valvular stenosis or regurgitation, and aneurysm), the double injection technique by providing a larger quantity of contrast medium and a faster bolus enhances the degree of opacification of the cardiovascular system; (3) bilateral superior venae cavae opacification and visualization of the degree of venous stenosis or occlusion when a mediastinal tumor is suspected are also disclosed by the double-barreled injection technique; and (4) by taking advantage of the 2 cannulas and performing angiocardiography via injection into both veins whenever it is planned to visualize the abdominal aorta and peripheral vascular system with the intravenous method, a superior study of the cardiovascular system will result because the bolus effect is enhanced by the double injections; dilution of the superior vena caval blood by the unopacified blood from the opposite innominate vein is also prevented.

SUMMARY AND CONCLUSIONS

In a series of 500 patients who have had bilateral simultaneous injections of concentrated contrast agents primarily for abdominal aortography and peripheral arteriography, 87 had contrast study of the cardiovascular system. Because a larger dose of contrast material and a faster bolus can be given with the double intravenous injection technique, improved cardiovascular visualization can be obtained in obese and heavily-built people. Similarly because of the larger dose of contrast agent, patients with cardiomegaly owing to heart failure, valvular disease or aneurysms will show a greater degree of opacification of their cardiovascular structures. The chief value of the bilateral injection technique in congenital heart disease is the demonstration of double superior venae cavae (and their respective courses into the cardiovascular system). Venous alterations produced by mediastinal tumors and estimation of benignancy or malignancy can also be learned from alteration of the great vein pattern.

Study of the cardiovascular system as part of a survey of peripheral vascular disease results in superior opacification and visualization of the cardiovascular structures because of enhancement of the bolus effect by the double intravenous injection technique and prevention of dilution of the contrast material by unopacified blood from the opposite innominate vein.

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ADDENDUM

Since this paper was submitted for publication, 155 additional patients have had bilateral intravenous injections without fatality. Of these, 42 had angiocardiography for cardiovascular disease.

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A TECHNIQUE USING CORONARY CINEARTERIOGRAPHY FOR SURGICAL AND PHYSIOLOGIC STUDIES IN DOGS*

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A PROJECT was designed primarily to study factors which are related to the formation of coronary collateral arterial channels. Coronary arteriography is an important phase of this study, which includes experimental methods for producing chronic coronary arterial insufficiency in dogs.

In these experiments, it was necessary to be able to demonstrate roentgenographically several types of abnormal situations in the coronary arterial system. These include: (a) narrowing or complete obstruction of a major artery, (b) a change in the ratio of the diameter of the circumflex artery in a postoperative dog as compared to the same dog preoperatively, (c) the presence of medium sized or large collateral channels between major arteries, and the direction of flow through them, (d) filling of a major vessel in a retrograde direction, and (e) abnormal rate of advancement of a column of contrast medium in one artery as compared to another.

This phase of the main project is being reported, because the technical information obtained may be useful to others doing experimental work. Also, the knowledge gained from study of the roentgen findings in these experimental preparations might later be helpful in interpretation of studies of human coronary arteries.

Several techniques are possible for coronary arteriography. These have been well described and reviewed in the literature.[4–8,12] Each method has specific advantages for specific purposes. Cinefluorography was chosen for the current work for several reasons. The rapid rate of exposure provided by filming at 30 frames per second appears to be necessary in order to obtain all of the information mentioned above, and this does not seem to be practical by any other technique. Other advantages of cinefluorography are the ability to fluoroscope before and during the positioning of the catheter, and immediately before filming. A short exposure time for each frame can be successfully achieved, and the film cost is much less than that of the large films for direct serial exposures.

Techniques employing cardiac arrest with or without roentgenographic magnification[1,3,10] give much more detail than cinefluorography. However, these methods were not satisfactory for the current project because of the need for demonstration of the flow of the opaque medium through coronary arteries with the heart beating. Furthermore, visualization of very small vessels was not necessary for the information desired. Direct filming with a non-arrest technique could be done with a rapid changer, if equipment such as the Dynapulse apparatus were used to provide a short exposure time. However, it is difficult to get enough frames per second with this type of apparatus.

The technical problems associated with coronary cinearteriography have been worked out, and consistently satisfactory results have been obtained.

EQUIPMENT AND ITS USE

The major items were made by the Picker X-Ray Corporation. A standard

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† Veterans Administration Clinical Investigator.

300 ma. generator was used with a half wave rectified circuit for 30 and 60 frames per second.

A Dunlee HRZ-9 x-ray tube was used with high speed rotation of 6,000 rpm. Although focal spots of 0.5 and 1.5 mm. were available in the tube, only the smaller one was utilized. The stationary grid had a six to one ratio with 60 lines per inch. The image amplification tube had an input phosphor 20 cm. in diameter. The minimum guaranteed brightness of such a tube is 1,000 times the input, and the minimum resolution is 40 line pairs per inch.

The 35 mm. camera was made for Picker by Photomechanisms. The camera motor was synchronized with the 60 cycle current source. It could be run at 60, 30, 15, or 7½ frames per second. The total exposure time was 1/120 of a second at 30 or 60 frames per second but the shape of the sine wave provided adequate power for film darkening for only about 1/200 of a second. The exposure time was approximately 1/30 and 1/15 of a second at 15 and 7½ frames per second, respectively.

An overframing lens was used so that the diameter of the tube corresponded to the diagonal of the frame on the film. This means that the entire area of the film was exposed.

There was a milliamperage selector switch on the apparatus to provide lower milliamperage when the apparatus was used for projects in which less contrast was desired. To demonstrate coronary arteries, the milliamperage was usually kept as high as possible in order to permit use of a low kilovoltage and obtain better contrast between the heart and coronary arteries. The indicated milliamperage was usually about 20. This means that the instantaneous milliamperage was about 80 at 30 frames per second, and about 40 at 60 frames per second.

There was a photoelectric cell in the system which automatically controlled the kilovoltage and kept the amount of light reaching the film near the desired level. The intensity of this light was indicated on a light meter in an arbitrary scale. The actual kilovoltage was not known but was believed to be between 70 and 110. Although it could be controlled by a manual dial, this was rarely done during the project.

The light meter was carefully observed even when the automatic kilovoltage adjustment was used in order to assure proper function of the apparatus. Although the scale was arbitrary, the relationship between the light meter reading for proper film darkening and the number of frames per second was demonstrated to be linear.

The position of the shutters made some difference in the light meter adjustment, and therefore was kept in a constant relative position for all calibrations and runs. Wires which could be seen in the viewing mirror were used for adjustment of the shutters. These wires corresponded to the edges of the frame of the film. Opening of the shutters wide enough to permit visualization of a complete circle of light in the mirror resulted in a little less resolution than was obtained when the shutters were closed to the wires.

The maximum permissible length of run was determined by calculations based on the heat capacity of the x-ray tube. Ten seconds was considered the maximum when an indicated milliamperage of 20 was being used.

The height of the image amplification and camera unit could be adjusted so that the distance from the table to the input phosphor was between about 11 and 24 inches. As roentgenologic magnification was found to improve detail when cardiac arrest was employed,[10] a study was made of the resolving power of the apparatus at various heights. Resolution was best in approximately midvertical position.

In routine practice, the apparatus was usually raised until the heart was magnified enough to fill the frame of the film. This resulted in a maximum area on the film per unit area of the heart. It also prevented the inclusion of much lung field, which would have influenced the light meter reading and affected the exposure adversely.

Most studies were done at 30 frames per second. This speed gave a combination of high contrast and short exposure time, and was used for all of the films reproduced as figures. Occasionally, 60 frames were taken per second.

Several types of film were tested. Eastman Kodak Cineflure gave good contrast but the grain was very large and its high speed was not necessary with the equipment being used (Fig. 1). Eastman Shellburst gave good contrast, but had moderate sized grain. Eastman Plus-X was preferred and was used for the majority of the studies. The grain size was minimal and the speed was adequate (Fig. 2). DuPont Superior No. 2 was tested, and was used extensively for another project with the same apparatus. It was not very satisfactory, because it frequently jammed in the magazine. These jams have been very rare with Eastman film.

A Vidicon television system was initially installed in the apparatus by Picker. It was not used because a better picture could be obtained from the mirrors, and because of instability. This apparatus was later removed.

A Smith-Picker 35 mm. processor was used for developing the film. Several developers were evaluated. X-ray developer gave good contrast, but resulted in very large grain in comparison to the other developers tested. Eastman Dektol gave good contrast but the grain still was a problem. Eastman D76 gave less contrast with little improvement in grain. Eastman D11 and D19 gave minimal grain size and good contrast on Plus-X film, but D19 gave more grain on Cineflure. Therefore, D11 was used for all of the coronary arteriograms herein reported. This developer is customarily employed in high contrast lithography and is quite readily available.

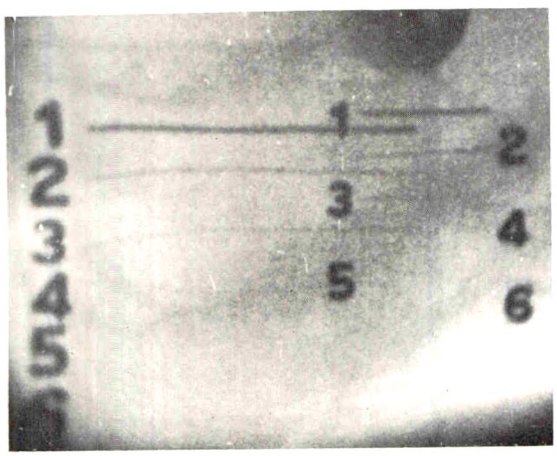


Fig. 1. Similar wire standards were placed above and below the chest of a dog in the lateral position. The image amplifier and camera unit were raised 22 inches above the table. The diameter of wires 1 through 4 is 900, 450, 300, and 200 μ, respectively. Eastman Cineflure film was used. Notice the grain size.

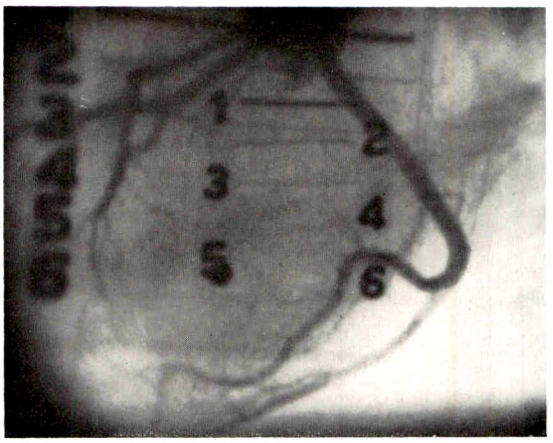


Fig. 2. The same technique as in Figure 1 was used, but with Eastman Plus-X film and with the left coronary artery injected with hypaque. The grain size is smaller than that in Figure 1 and arteries as small as wire No. 3 can be seen.

The usual developing time was 6 minutes, but was sometimes increased to 7 or 8 minutes. All developing was done at 68°F.

Reduction printing to 16 mm. was done with an apparatus made by Uhler Cine Machine Company.* The light scale was arbitrary and had to be determined experimentally for each scene. The 16 mm. film was all developed in D11 at 68°F. for six minutes.

Using this technique, Eastman Fine Grain Duplicating Positive film gave the most faithful reversal of the negative. Eastman Fine Grain Release Positive film

* Purchased from Picker X-Ray Corporation, 1707 E. 7th Street, Charlotte, North Carolina.

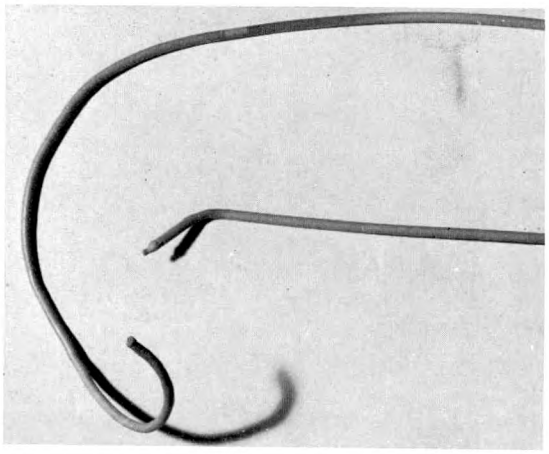


FIG. 3. The yellow radiopaque polyethylene catheters that were used for injection. The one with the bend near the end is for the carotid approach. Note the sealed end and the single hole near the tip. The other catheter is for the femoral approach. The three dimensional curve (note shadow) was designed to approximate the shape of the aortic arch.

increased the contrast somewhat, and gave the most satisfactory 16 mm. prints of coronary arteries. Eastman High Contrast Positive film gave more contrast, but this was undesirable and sometimes failed to demonstrate the coronary arteries in both the lighter and darker areas of the heart shadow.

TECHNIQUE OF INJECTION

The dogs were anesthetized with nembutal, and an endotracheal tube was inserted. No respirator or oxygen supply was used.

The catheters* were made from yellow Ödman radiopaque polyethylene with an outside diameter of about 0.110 inches. One end of each catheter was closed. A single hole about 2 mm. in diameter was cut about 3 mm. from the closed end. The hole was located so that the jet would point toward the orifice of the left coronary artery.

The carotid or femoral vessel was exposed through a small transverse incision, using clean but not aseptic technique. The wounds were left open and without exception drained effectively and healed satisfactorily within one to two weeks with no evidence of difficulty from infection.

All injection was done into the left sinus of Valsalva. A satisfactory position was obtained most easily through the carotid approach using a catheter which had a bend about 2 cm. from its end. This curve was found to be difficult to introduce through the femoral artery until a catheter was fashioned which approximated the shape of the aortic arch (Fig. 3).

Each dog was first placed in a very steep left anterior oblique position that approximated the posteroanterior position. The catheter could be inserted into the left coronary sinus under fluoroscopic control without difficulty. Preliminary studies showed that injection into the aorta above the cusp wasted contrast medium and offered no advantages.

Injection directly into the coronary artery would have given excellent contrast, as was shown by the splendid work of Sones and Shirey,[9] West *et al.*,[13] and Tapia *et al.*[11] This was not done because information on the relative rates of flow in the anterior descending and circumflex arteries was desired, and the presence of a catheter in one or the other branch might have altered these proportions.

Correct position of the catheter within the left sinus of Valsalva was noted to be very important. This location was found by slow manual injection of 1 to 3 cc. of hypaque under fluoroscopy. When the catheter was correctly positioned, all of the hypaque which could be seen in the image intensifier viewing mirror went into the coronary arteries and none was washed up the ascending aorta. Any other position of the catheter was likely to result in the loss of a large percentage of the medium.

Several types of injectors were tried. These included a hand injector, a manually operated Gidlund lever apparatus made by Elema Jarnh, and a Zimmerman Pemco† pneumatic injector. The pneumatic apparatus was greatly preferred be-

* Purchased from Schick X-Ray Co., Inc., 205 West Wacker Drive, Chicago 6, Illinois.

† Purchased from Pemco, Inc., 5663 Brecksville Road, Cleveland 31, Ohio.

cause of its consistency, reliability, and ease of operation. Although more pressure could have been used, 20 pounds per square inch was found to be most satisfactory. This injected 15 cc. of warm 90 per cent hypaque in about $2\frac{1}{2}$ seconds, which covered about six cardiac cycles. The apparatus was kept warm by an infrared heat lamp.

Warm 90 per cent hypaque was used for all but a few of the studies. It was found to be entirely satisfactory. Ditriokon could be injected more rapidly because of its lower viscosity, but this was of no advantage in these studies. More medium spilled over into the aorta and the contrast of the arteries was not as great.

The technique described was found to be entirely practical for large scale work. Oblique and lateral projections of each dog were usually secured at the rate of one dog every 15 or 20 minutes. Coronary arteriograms were obtained on as many as four different occasions on some dogs by using both carotid and both femoral arteries.

RESULTS

Tests of the resolving power of the apparatus were done with a wire standard at various degrees of roentgenologic magnification. A steel wire 300 μ in diameter could be consistently demonstrated on the viewing screen (Fig. 1 and 2). In the mid-



Fig. 4. Arteriogram of a normal dog in the left anterior oblique position. The origin of the anterior descending artery can be seen beside the left sinus of Valsalva.

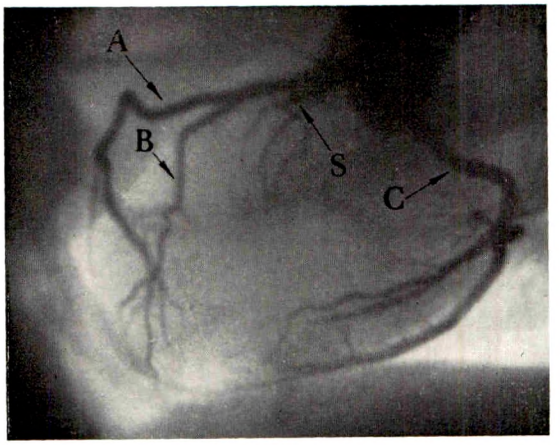


Fig. 5. Lateral projection of the same dog as in Figure 4. This is the first frame in which the anterior descending artery was maximally filled. A—anterior descending artery, B—branch of the anterior descending artery, C—circumflex artery, S—septal branch.

range of the available roentgenologic magnification, a 200 μ wire could be seen rather vaguely.

Comparison with these wire standards and careful calculation of the degree of magnification showed that coronary arter-

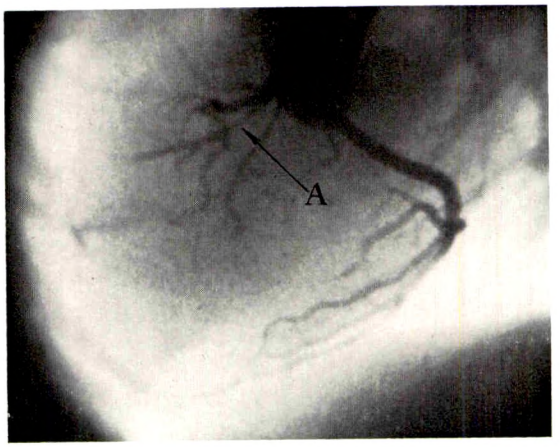


Fig. 6. Arteriogram of the same dog as in Figures 4 and 5 taken eight days after surgical narrowing of the proximal anterior descending artery. The area of narrowing (A) can be seen just under the septal branch. This is the first frame in which the circumflex artery is filled the same percentage of the distance from the aortic valve to the apex of the heart as it was in Figure 5. The anterior descending artery has not filled very far distally in this frame.

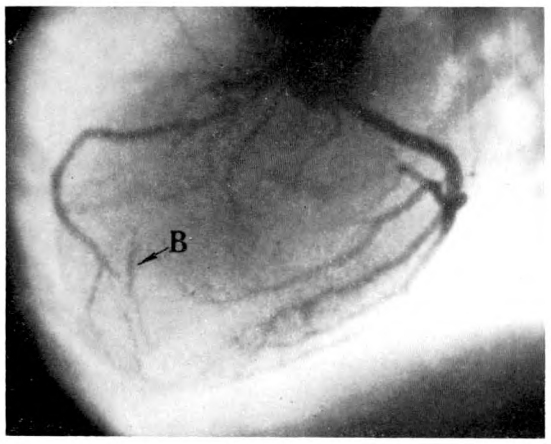


FIG. 7. Several cardiac cycles later in the same series as Figure 6. Maximal filling of the coronary arteries has occurred. The filling defect in the proximal anterior descending artery can still be seen. The proximal end of the large branch (B in Figure 5) was sacrificed at surgery for pressure measurements. The distal end (B) is partly filled in this frame through collateral circulation. The diameter of the distal anterior descending artery in relation to that of the circumflex artery is considerably less than it was in the preoperative arteriogram.

ies were consistently demonstrated down to a diameter of about 500 μ. Vessels as small as 250 μ were sometimes seen. Very little vignetting was observed, and the quality of the pictures in general was consistent and satisfactory.

Three hundred and eight injections were

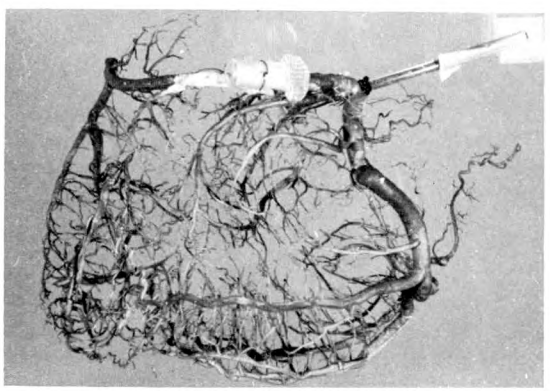


FIG. 8. A vinylite corrosion cast from the heart of the dog shown in Figures 4–7, viewed as in the lateral projection. The dog was sacrificed about two months postoperatively. The smallest vessels filled are about 100 μ in diameter. Compare with Figure 5.

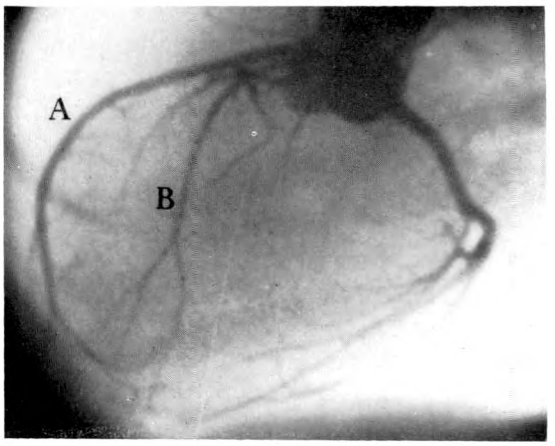


FIG. 9. The preoperative arteriogram of another dog in the lateral position. A—anterior descending artery, B—a large branch that will be used for measurement of pressure at surgery.

done on 128 occasions on 74 dogs. Consistent success was obtained after the first 77 injections.

Examples of typical findings are shown in Figures 4–11. Normal left coronary arteries were visualized. Complete obstruction of the anterior descending artery or

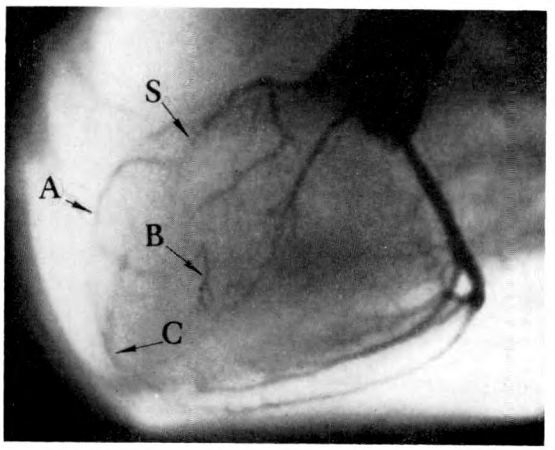


FIG. 10. Arteriogram of the same dog as in Figure 8 taken two months after surgery. The anterior descending artery is almost completely occluded. There is a collateral channel from the anterior branch of the septal artery (S) to the anterior descending artery (A). There is another collateral from the left marginal artery which passes around the apex (C) to the distal end of the anterior descending artery. The distal end of the branch used for measurement of pressure (B) has filled from a collateral from a branch of the anterior descending artery proximal to the constriction.

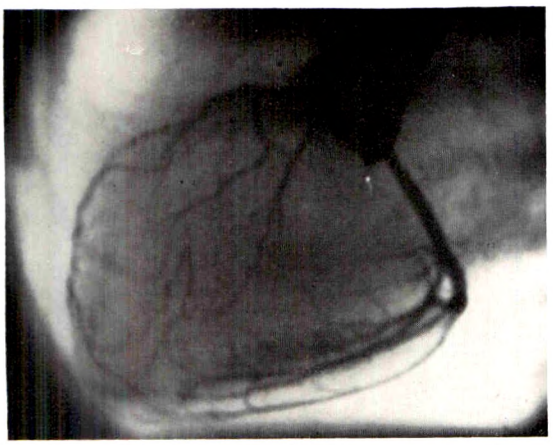


Fig. 11. Several cardiac cycles after Figure 10. The anterior descending artery is now largely filled through collateral channels but appears small and somewhat irregular. If Figure 10 were not available, it would be impossible to tell from which direction various portions of the anterior descending artery had filled.

narrowing of its proximal portion was demonstrated. Collateral channels were frequently seen. The direction of flow through them and retrograde filling of the anterior descending artery distal to an obstruction was observed. After the proximal portion of the anterior descending artery had been narrowed surgically, the distal portion was frequently found to be smaller in relation to the circumflex artery than it had been in the preoperative arteriogram. A decrease in the velocity of movement through this artery in comparison to the circumflex artery was also found in some of the postoperative dogs.

During some of the early studies, several repeat injections were made in some dogs. The maximum tolerated dose appeared to be about 100 cc. of hypaque in multiple injections of 15 to 25 cc. Dogs receiving more usually died 24 to 48 hours after arteriography, and the probable cause of death was renal damage. The dogs weighed between 25 and 40 pounds, and this dose is somewhat larger than the LD_{50} of 3 cc./kg. recently reported.[2] The difference is probably explained by the fact that the total dose in the current series was divided instead of injected at one time.

Radiation exposure of personnel was found to be about 20 mr per month above background, as indicated by detectors placed outside of the lead aprons.

SUMMARY

Coronary cinearteriography was used for surgical and physiologic studies in dogs. A 20 cm. Picker image amplification tube and a 35 mm. camera were employed.

All injections were done into the left sinus of Valsalva. Cardiac arrest was not resorted to. Most filming was performed at 30 frames per second on Eastman Plus-X film.

The study successfully demonstrated (a) surgical narrowing or complete obstruction of a major coronary artery, (b) a change in the ratio of the diameter of the distal anterior descending artery to the diameter of the circumflex artery in a postoperative dog as compared to the same dog preoperatively, (c) the presence of medium sized or large collateral channels between major arteries, and the direction of flow through them, (d) filling of a major vessel in a retrograde direction, and (e) abnormal rate of advancement of a column of contrast medium in one artery as compared to another.

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VOL. 88, NO. 1

THE ROENTGENOGRAPHIC MANIFESTATIONS OF AORTIC STENOSIS AND AORTIC VALVULAR INSUFFICIENCY*

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DUE to the improvement in cardiovascular surgery, it is now possible to repair many aortic valvular and paravalvular lesions. Recognition of these abnormalities has therefore gained considerable importance. While the classic roentgenographic appearance of patients with aortic stenosis or aortic valvular insufficiency is well known, the reliability of certain signs and their significance in differential diagnosis is not generally appreciated. It thus seemed appropriate to report the roentgenographic manifestations in a large group of patients with proven abnormalities of the aortic valvular and paravalvular area.

MATERIAL

Eighty-nine cases were available for study (Table 1). Only those with proven aortic stenosis and aortic valvular insufficiency studied at Indiana University Medical Center during the last 4 years were included. All patients with congenital and most patients with acquired lesions had left heart catheterization followed by cinecardioangiography. The majority of the patients had surgical confirmation of the diagnosis.

This group includes 42 patients with congenital and 47 with acquired lesions. The abnormality was considered congenital if a cardiac defect was known to be present prior to 5 years of age and there was no history of rheumatic fever. The group classified as acquired had no known heart disease in infancy.

The high percentage of congenital lesions in this series probably reflects the larger volume of a busy pediatric cardiovascular service at this institution. Congenital aortic valvular stenosis was more common than subvalvular stenosis in a ratio of 23 to 14 cases, respectively. Eighty-five per cent of congenital and 70 per cent of acquired lesions were in males. The mean age of patients with congenital valvular stenosis and those with subvalvular stenosis was essentially the same, 10 and 13 years, respectively. The mean age of patients with acquired aortic stenosis was 44, and with aortic valvular stenosis and insufficiency 43. It was of interest that those patients with predominant aortic valvular insufficiency had a mean age of 28. This probably reflects the more serious nature of this abnormality in the cases studied.

CLASSIFICATION

Stenosis in the region of the aortic valve can best be divided into supravalvular, valvular and subvalvular.

Supravalvular stenosis is quite rare. Less than 30 cases have been reported in the literature.[9,10,12,24,26,27,32] Three of these cases had associated stigmata of Marfan's syndrome.[9,27] The most common supravalvular lesion is a narrowing or coarctation just above the ostia of the coronary arteries. Thickening of the plicae of the sinuses of Valsalva may form an encircling ridge of fibrous tissue. The free margin of one or more valve leaflets may be adherent to the aortic wall resulting in associated aortic valvular insufficiency. A less common cause

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† Public Health Research Fellow, National Heart Institute.

TABLE I

MATERIAL

	Acquired Aortic Insufficiency	Acquired Aortic Stenosis and Aortic Insufficiency	Acquired Aortic Stenosis	Congenital Valvular Aortic Stenosis	Congenital Subaortic Stenosis
No. of Cases	13	8	26	23	14
Age Range (yr.)	12–51	32–61	24–60	4½–33	2½–36
Average Age	28	43	44	10	12.7
Sex F	4 (31%)	1 (12.5%)	9 (35%)	2 (9%)	4 (29%)
M	9 (69%)	7 (87.5%)	17 (65%)	21 (91%)	10 (71%)

5 cases not tabulated above include supravalvular stenosis—1; congenital aortic stenosis and insufficiency—3; congenital aortic insufficiency—1.

of supravalvular stenosis is an obstructing membrane inserting into the aortic wall at the level of the commissures.

Another lesion which has been classified with supravalvular stenosis is a fibrous band extending across the aortic lumen at the level of the aortic valve commissures.[7] This condition may be associated with atrophy of the left coronary cusp resulting in aortic insufficiency.[34] More commonly, this lesion is not a true obstruction and is usually an incidental autopsy finding of no clinical significance.

The etiology of valvular aortic stenosis is frequently obscure. When the abnormality is detected prior to 5 years of age, and there is no history of rheumatic fever, it is reasonable to assume that the lesion is congenital. The aortic valve originates from division of the four bulbar cushions and faulty division may result in aortic stenosis. Fetal endocarditis has been described as an etiologic agent,[8] but there is little evidence to support this view. In older patients with valvular aortic stenosis, the etiology was frequently not known. No doubt some of these cases represented congenital aortic stenosis undetected during childhood. A definite history of rheumatic fever could be obtained in less than one-half of these patients. The rheumatic etiology of pure aortic stenosis has been questioned by Kiloh,[21] who found no evidence of rheumatic fever in 27 patients with pure aortic stenosis. In contradistinction, he reported a history of rheumatic fever in 60 per cent of patients with aortic valvular stenosis and insufficiency. Bland and Jones[2] in their 20 year follow-up of 1,000 children with rheumatic fever found only 2 cases of pure aortic stenosis and 27 cases of aortic stenosis and insufficiency. Perhaps the strongest support for a rheumatic basis of aortic stenosis is the study of Karsner and Koletsky.[17] These authors investigated 200 cases of aortic valvular stenosis coming to autopsy. They reported that only 2 per cent were not on a rheumatic basis. The actual etiology is not a vital factor since the pathologic physiology and anatomy are similar in both acquired and congenital aortic valvular stenosis. A difference in the clinical course of these two types of aortic valvular stenosis has not been clearly shown. By means of left heart cinecardioangiography, it was noted that the stenotic valve leaflets of children were usually quite mobile and dome-shaped during ventricular systole. In contradistinction, the aortic valves in adult patients were thickened and frequently had little movement.

Until recently, stenotic lesions in the subvalvular area were considered to be essentially benign and extremely rare. There were less than 50 cases reported in the literature prior to 1950. More recent reports have

indicated an increase in the frequency of recognition of these lesions. Brock and Fleming[5,6] reported 12 cases diagnosed during life and suggested that the subaortic fibrous lesion may be as common as the valvular. Hancock,[14] reporting a series of patients, stated that severe aortic stenosis is subvalvular in 15 per cent of all cases and in nearly 50 per cent of those under 25 years of age. The embryology of subvalvular aortic stenosis was described by Keith.[18–20] It was his opinion that subvalvular stenosis represented lack of embryonic maturation with persistence of the bulbus cordis. In the three chambered vertebrate heart, the bulbus cordis functions as a separate muscular chamber between the common ventricle and the aorta. In the human fetus the major portion of the bulbus cordis is incorporated into the infundibulum of the right ventricle. Normally, there is total atrophy of the bulbus portion of the left heart. Arrest of growth of the bulbus in the right heart leads to infundibular pulmonic stenosis; arrest of atrophy of the bulbus in the left heart leads to subaortic stenosis. A remnant of fibrous tissue may persist as a subaortic fibrous band. This fibrous ring is usually 5–22 mm. below the aortic cusps and encircles the aortic conus. Arrest of atrophy of the bulbus cordis may occur while it is still a contractile muscular structure so that it remains as an intrinsic functional portion of the interventricular septum. Obstruction of the outflow tract of the left ventricle would thus result during ventricular systole from apposition of the hypertrophied septum and the left ventricular wall. It is of interest that 2 of our cases of subvalvular stenosis secondary to hypertrophy of the interventricular septum had associated pulmonary infundibular stenosis.[22] Teare[33] presented the autopsy description of the hearts of 8 patients ranging in age from 14 to 45 years who had hypertrophic subaortic stenosis. Seven of these patients died suddenly and the eighth died after an attempted mitral valvulotomy. At postmortem examination, there was diffuse hypertrophy of the interventricular septum in all patients with some extension of the process to the anterior wall of the left ventricle in several instances. He considered the abnormality to be developmental in origin. The systolic contraction of an area of diffuse muscular hypertrophy in the ventricular outflow tract as the cause of some cases of subvalvular aortic stenosis has been reported by a number of authors.[1,3–6,14,22,2,529,33] This condition has been called pseudo-aortic stenosis, functional aortic stenosis or idiopathic hypertrophic subaortic stenosis. The latter seems the most descriptive term. It is important to differentiate this group of cases from the more common fibrous ring in the outflow tract of the left ventricle since surgical correction of idiopathic hypertrophic subaortic stenosis is not possible. Left heart angiocardiography is necessary to make this differentiation.

ROENTGENOGRAPHIC MANIFESTATIONS

CARDIAC SIZE

In aortic stenosis of either the valvular or subvalvular area, the heart was usually not enlarged (Table II). Over one-half had a cardiothoracic ratio of below 0.5. Cardiac

TABLE II

CARDIOTHORACIC RATIO

	Acquired Aortic Insufficiency	Acquired Aortic Stenosis and Aortic Insufficiency	Acquired Aortic Stenosis	Congenital Valvular Aortic Stenosis	Congenital Subaortic Stenosis
Range	.41–.66	.45–.64	.43–.69	.37–.65	.44–.66
Average	.57	.54	.51	.48	.51

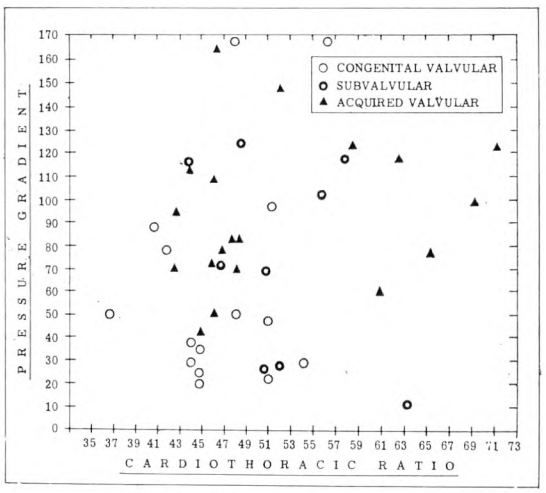


FIG. 1. Comparison of cardiac size and severity of aortic stenosis.

size was of no value in differentiating aortic valvular from subvalvular stenosis. The mean cardiothoracic ratio of patients with congenital valvular stenosis was .48; with congenital subvalvular stenosis .51; and with acquired valvular stenosis .51. No correlation between the cardiothoracic ratio and the pressure gradient existed (Fig. 1).

As a general rule, the heart size was larger when insufficiency was combined with stenosis (mean cardiothoracic ratio .54) and was largest in the group with predominant aortic insufficiency (mean cardiothoracic ratio .57).

LEFT VENTRICLE

In spite of the fact that the heart was frequently not enlarged in aortic stenosis, it was usually abnormal in configuration secondary to concentric hypertrophy of the left ventricle (Table III), (Fig. 2, *A* and *B*). On angiocardiograms it was noted that the left ventricular cavity was usually small in pure aortic stenosis unless left ventricular failure was present. With aortic insufficiency, there was a marked increase in the diastolic volume of the left ventricle secondary to diastolic filling through both the aortic and mitral valves.

Concentric hypertrophy of the left ventricle was roentgenographically manifest by a circumscribed convex bulging of the lower one-third of the left cardiac border. The

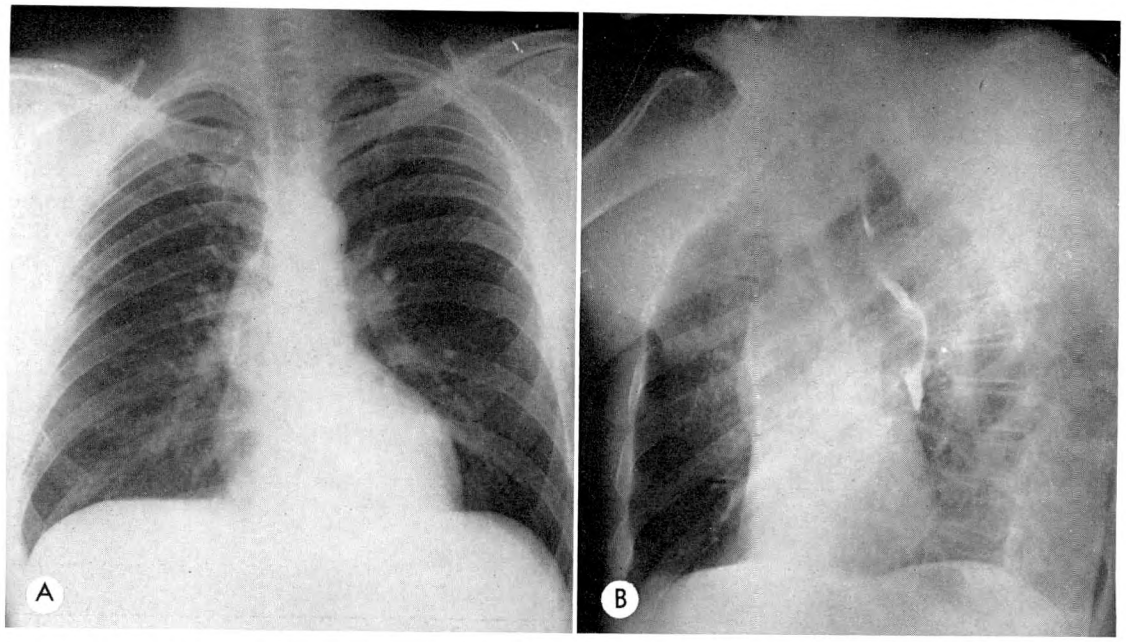


FIG. 2. (*A* and *B*) Acquired aortic valvular stenosis. Note the appearance of concentric hypertrophy of the left ventricle. The apex is not displaced downward and posteriorly in the left anterior oblique view. The ascending aorta is dilated and the aortic knob is normal.

Table III

LEFT VENTRICULAR SIZE

Extent of Hypertrophy	Acquired Aortic Insufficiency		Acquired Aortic Stenosis and Aortic Insufficiency		Acquired Aortic Stenosis		Congenital Valvular Aortic Stenosis		Congenital Subaortic Stenosis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Casse	Per Cent
+	1	8	0	0	3	12	4	17	2	14
++	6	46	4	50	20	77	16	8	8	57
+++	6	46	4	50	2	8	2	9	2	14
Total	13	100	8	100	25	97	22	96	10	85

most lateral point of this curve lies at a higher level above the diaphragm than in normal hearts. The cardiac waist may appear relatively normal. At times this configuration is difficult to distinguish from hypertrophy of the right ventricle.

The apex was elongated posteriorly and inferiorly in only 5 of 63 cases of aortic stenosis. In these instances, left ventricular dilatation secondary to left ventricular failure was present. In contradistinction, in aortic insufficiency, posterior and inferior elongation of the cardiac apex was the rule —being present in 11 of 13 cases (Table IV). Only with dilatation of the left ventricle was the conspicuous narrowing of the cardiac waist visible.

Elongation of the apex thus appears to be secondary to dilatation of the left ventricular cavity and is an important differential point between aortic stenosis and aortic insufficiency (Fig. 3, *A* and *B*).

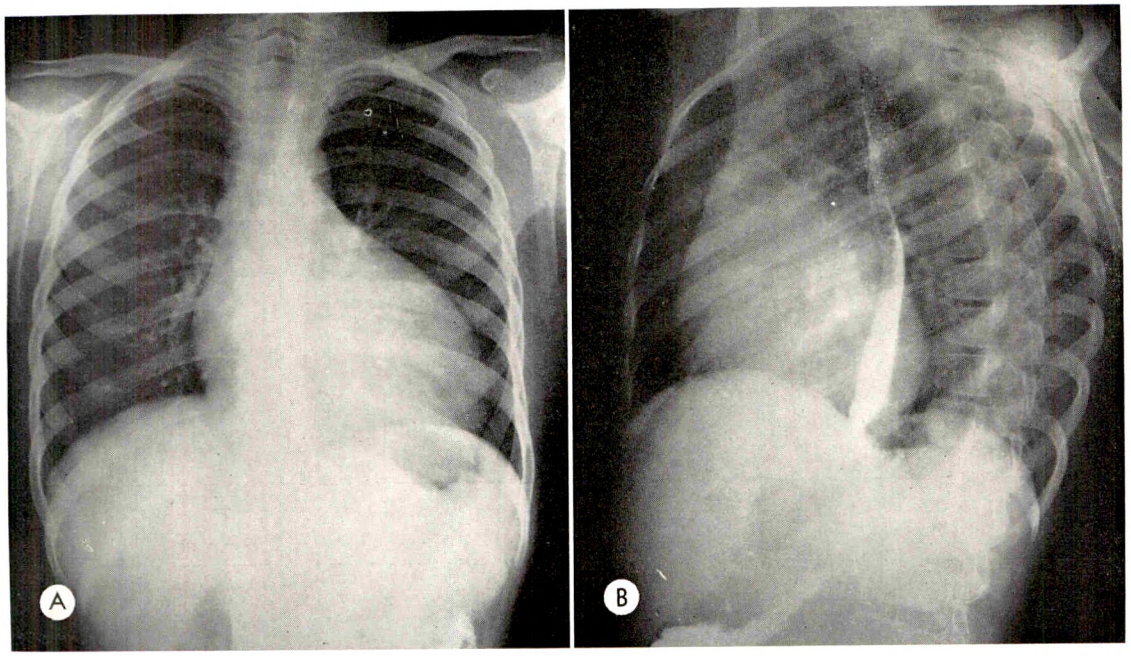


Fig. 3. (*A* and *B*) Acquired aortic valvular insufficiency. The left ventricle is dilated. The cardiac apex is displaced downward and posteriorly. The ascending aorta is not dilated.

TABLE IV

ELONGATION OF THE CARDIAC APEX

	Acquired Aortic Insufficiency		Acquired Aortic Stenosis and Aortic Insufficiency		Acquired Aortic Stenosis		Congenital Valvular Aortic Stenosis		Congenital Subaortic Stenosis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
Apex Down	11	85	2	25	1	4	4	17	0	0

LEFT ATRIUM

The left atrium was frequently enlarged (Table v). It was commonly minimal, but in 29 per cent of patients with subvalvular stenosis, left atrial enlargement was moderate. In only 4 per cent of patients with congenital valvular stenosis was the left atrium more than minimally enlarged. The larger left atrial size in congenital subvalvular stenosis may be secondary to distortion of the mitral valve by the subaortic muscular hypertrophy or the subvalvular fibrous ring. The left atrium was moderately to markedly enlarged in 50 per cent of patients with aortic stenosis and insufficiency and in 16 per cent of patients with predominant aortic insufficiency. Left atrial enlargement in these cases was usually associated with chronic left ventricular failure with an elevated end diastolic pressure (Fig. 4, *A* and *B*). This pressure would be reflected by an elevated left atrial pressure causing this chamber to enlarge.

PULMONARY HYPERTENSION

Pulmonary hypertension was not uncommon in patients with acquired aortic valvular disease.[28] In 3 cases, this was quite marked and the presenting symptoms were those of right ventricular failure. These cases were frequently misdiagnosed as double valvular lesions. If specific aortic signs are not present, the mistaken diagnosis of mitral insufficiency may be made (Fig. 5). Pulmonary hypertension is the result of elevation of the left atrial pressure secondary to chronic failure of the left ventricle. A reflex constriction of the medium sized pulmonary arteries may cause further elevation of the pressure in the pulmonary artery. Eventually intimal hyperplasia and fibrosis of the pulmonary vessels leads to irreversible changes.

AORTA

Dilatation of the ascending aorta is a valuable sign of aortic stenosis (Table VI).

TABLE V

LEFT ATRIAL SIZE

Extent of Enlargement	Acquired Aortic Insufficiency		Acquired Aortic Stenosis and Aortic Insufficiency		Acquired Aortic Stenosis		Congenital Valvular Aortic Stenosis		Congenital Subaortic Stenosis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
+	4	31	2	25	6	23	8	35	2	14
++	1	8	4	50	3	12	0	0	4	29
+++	1	8	0	0	0	—	0	0	0	0
Total	6	47	6	75	9	35	8	35	6	43

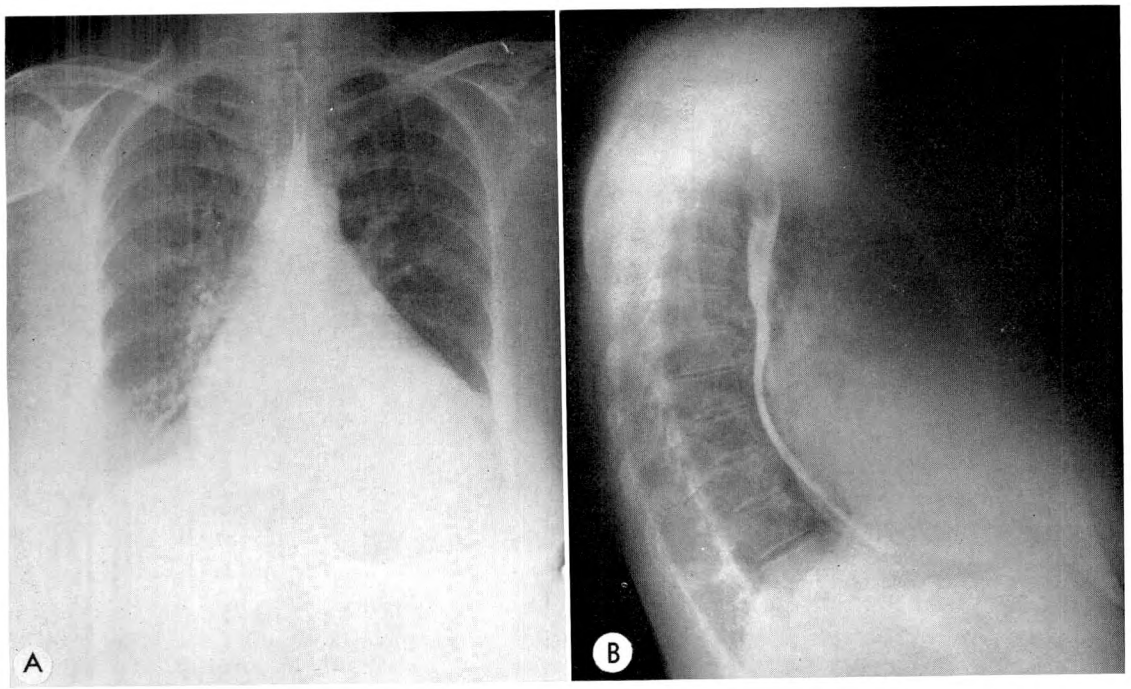


FIG. 4. (*A* and *B*) Aortic stenosis with left ventricular failure and enlargement of the left atrium. The misdiagnosis of mitral insufficiency may be made.

This was first described by Chevers[11] in 1842. A number of theories have been proposed to explain this phenomenon.[13,15,16,30] Most authors agree that an important factor is the high frequency vibrations that are set up in the vessel wall by the eddy currents produced by stenosis and result in structural alterations of the tunica media.

Poststenotic dilatation was present in 71 per cent of patients with subvalvular stenosis, 74 per cent with congenital valvular stenosis and 84 per cent with acquired valvular stenosis. There was no correlation in the degree of poststenotic dilatation and the severity of the aortic stenosis. Poststenotic dilatation was not a good differential point between congenital valvular and subvalvular stenosis (Fig. 6, *A* and *B*). It was of interest that neither patient with hypertrophic subvalvular stenosis had sig-

TABLE VI

DILATATION OF ASCENDING AORTA

Extent of Dilatation	Acquired Aortic Insufficiency		Acquired Aortic Stenosis and Aortic Insufficiency		Acquired Aortic Stenosis		Congenital Valvular Aortic Stenosis		Congenital Subaortic Stenosis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
+	4	46	4	50	4	15	6	26	3	21
++	3	8	3	37·5	13	50	10	44	3	29
+++	0	0	0	0	5	19	1	4	3	21
Total	7	54	7	87·5	22	84	17	74	10	71

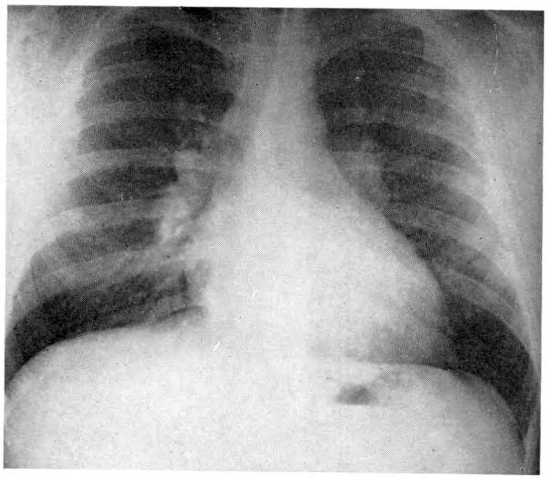


FIG. 5. Aortic insufficiency with pulmonary hypertension.

nificant poststenotic dilatation of the aorta (Fig. 7, *A* and *B*). The lack of poststenotic dilatation of the aorta in a child with aortic stenosis should make one consider this condition of supravalvular stenosis (Fig. 8). On angiocardiograms it was noted that the dilatation usually ended at the innominate artery. On plain roentgenograms, the knob was slightly prominent in 46 per cent of patients with acquired aortic stenosis, 44 per cent with congenital valvular stenosis and 7 per cent with congenital subvalvular stenosis. In only 15 per cent of patients with acquired aortic stenosis was it moderately enlarged and in no cases of congenital aortic valvular or subvalvular stenosis was this present. The lack of significant enlargement of the aortic knob is therefore valuable in differentiating dilatation of the ascending limb caused by aortic stenosis from aortic ectasia seen in elderly people.

The ascending aorta was enlarged in 54 per cent of patients with predominant aortic insufficiency. It was considered slight in 46 per cent and moderate in 8 per cent and in no instance was marked dilatation noted. This series did not include patients with luetic aortic insufficiency or Marfan's syndrome. It would thus appear that moderate to marked dilatation of the ascending aorta in a patient with aortic insufficiency would indicate significant associated aortic stenosis unless luetic aortitis or Marfan's syndrome was present.

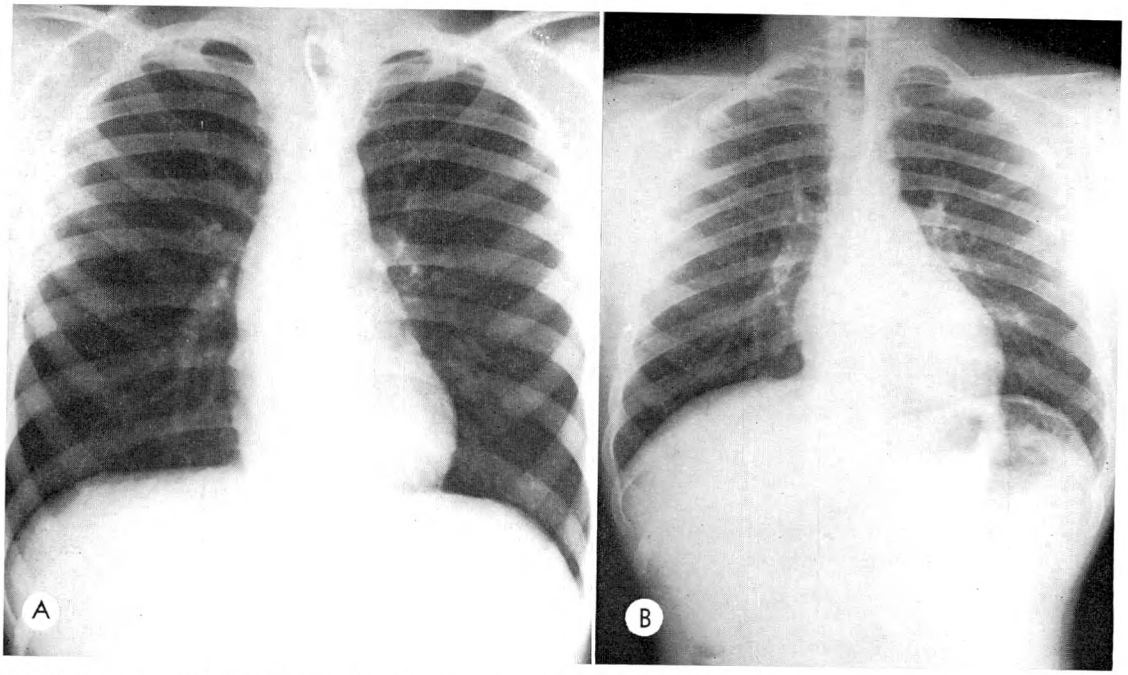


FIG. 6. (*A* and *B*) Congenital aortic valvular stenosis. Note the cardiac configuration of concentric hypertrophy of the left ventricle. The ascending aorta is dilated in *A* and normal in *B*. Dilatation of the ascending aorta was present in 74 per cent of patients with congenital aortic valvular stenosis.

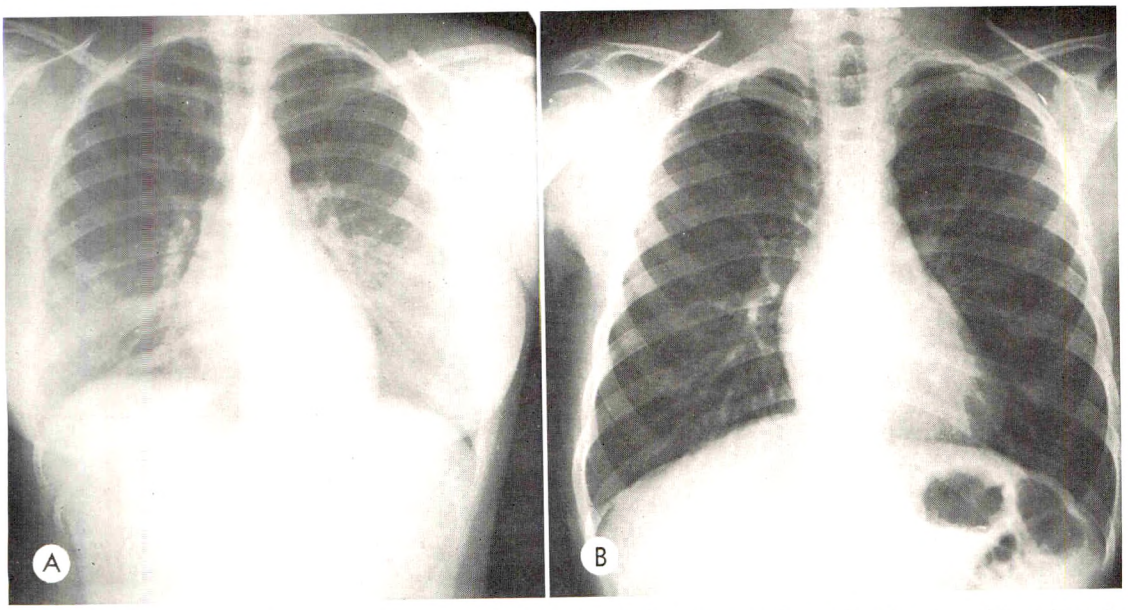


Fig. 7. Congenital subvalvular aortic stenosis. (*A*) Subvalvular fibrous ring. (*B*) Hypertrophic subaortic stenosis. The ascending aorta was dilated in 71 per cent of patients with subaortic stenosis. It was normal in both cases of the hypertrophic type.

Fluoroscopy proved to be of considerable value in the evaluation of the aorta. In patients with aortic stenosis, increased pulsation of the ascending aorta was usually present. It may occasionally be absent in patients with very severe aortic stenosis. In aortic insufficiency, increased pulsation of the entire aortic arch has consistently been noted. This fluoroscopic sign was an important differential point in evaluating aortic stenosis and insufficiency. Apparent increased pulsation of the entire aortic arch may occasionally be seen in patients with a slow ventricular rate secondary to heart block or athletic conditioning. The marked pulsation is apparently the result of increased stroke volume of the left ventricle. It is therefore important not to over-evaluate aortic pulsation in patients with a slow ventricular rate.

Valvular Calcification

Aortic valvular calcification is a valuable diagnostic sign (Table VII). It was present in 85 per cent of patients with acquired aortic stenosis. Of 63 patients of all ages with congenital or acquired valvular stenosis, valvular calcification was noted in 34. In only one of 33 patients below the age group of 20 years could valvular calcification be seen. It was noted in 3 of 5 patients

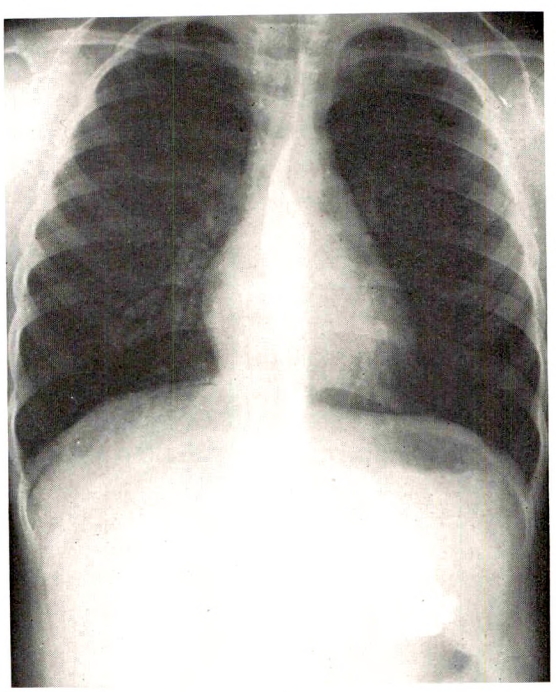


Fig. 8. Supravalvular aortic stenosis. Note the lack of dilatation of the ascending aorta. There are no specific aortic signs.

TABLE VII

CALCIFICATION OF AORTIC VALVE

Extent of Calcification	Acquired Aortic Insufficiency		Acquired Aortic Stenosis and Aortic Insufficiency		Acquired Aortic Stenosis		Congenital Valvular Aortic Stenosis		Congenital Subaortic Stenosis	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
+	2	15	2	25	3	12	0	0	1	7
++	1	8	2	25	13	50	1	4	0	0
+++	0	0	3	37.5	6	23	0	0	0	0
Total	3	23	7	87.5	22	85	1	4	1	7

of age 20–30, 6 of 8 patients of age 30–40, 6 of 8 patients of age 40–50 and 8 of 9 patients of age 50–60. Fluoroscopy is valuable in the diagnosis of aortic valvular calcification. Prior to the routine use of image amplification, laminagraphy was also utilized. This was discontinued after a comparison study of laminagraphy and image intensified fluoroscopy revealed the latter to be more accurate. The calcified valve can usually be demonstrated on the films but at times it is difficult to differentiate from calcification within the lung.

Our fluoroscopic technique is first to search for calcium with the patient in the right anterior oblique position. If no calcium is seen, the search for calcium is discontinued. Upon visualization of valvular calcification, the patient is turned into the left anterior oblique position to more widely separate the mitral and aortic valves. As described by Sosman and Wosika,[81] in the left anterior oblique view the aortic valve is located in the upper one-half of the middle one-third of the heart while the mitral valve is located in the posterior one-third of the lower one-half of the heart. On the posteroanterior view, the aortic valve is superimposed on the spine. One rarely demonstrates a calcified aortic valve on the posteroanterior view and calcium noted in this view is probably in the mitral valve.

SPECIAL TECHNIQUES

Since the advent of corrective surgery for aortic valvular and paravalvular lesions, specific information is needed which cannot be determined with routine roentgenographic procedures. In patients with congenital aortic stenosis, it is important to differentiate supravalvular, valvular and subvalvular lesions. The appearance of the aortic valve—whether it is fixed or mobile—and the degree of deformity can be best determined by left heart cinecardioangiography. One may differentiate a subvalvular fibrous ring from subaortic hypertrophic stenosis in which an abnormal muscular mass obstructs the outflow tract of the left ventricle. This is important since in the former, the lesion may be correctable while in the latter surgery is contraindicated. In acquired and congenital aortic valvular stenosis, the degree of associated valvular insufficiency and the presence or absence of concomitant mitral disease can be evaluated. If aortic valvular replacement is considered, it is important that the coronary arteries be evaluated. If moderate to severe disease is present, it may be impossible to adequately perfuse the coronary arteries during the surgical procedure. The gradient across the stenotic valve is important in evaluating the severity of the disease. Routine roentgenographic and clinical findings including electrocardiography are notoriously poor in evaluating severity. Because of these factors, it has been our policy to perform left heart catheterization and cinecardioangiography on all patients with sus-

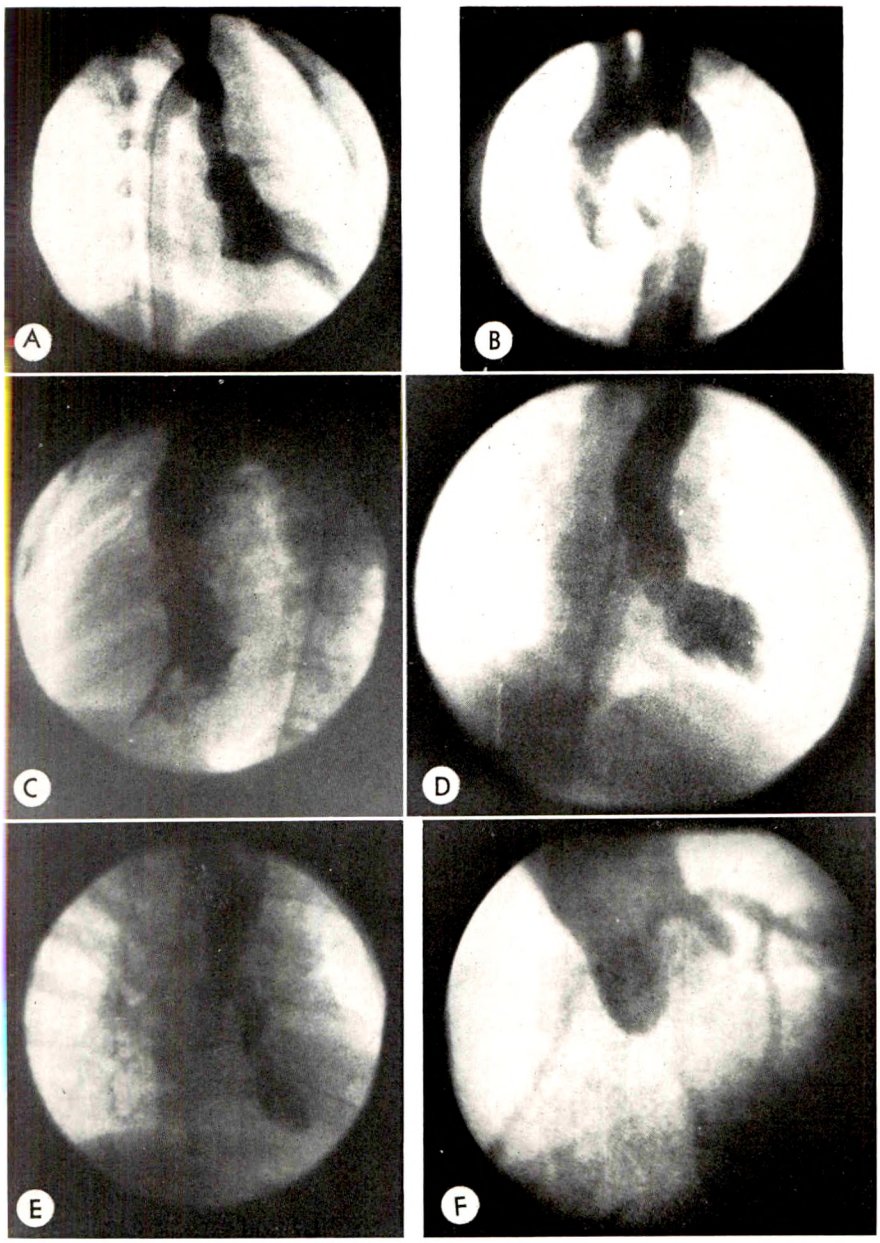


Fig. 9. Enlargements of 16 mm. cinecardioangiograms demonstrating various abnormalities of the aortis valvular and paravalvular areas. (*A*) Supravalvular aortic stenosis. (*B*) Congenital aortic valvular stenosis—aortic injection. (*C*) Congenital aortic valvular stenosis—left ventricular injection. (*D*) Subvalvular fibrous ring. (*E*) Hypertrophic subvalvular stenosis. (*F*) Acquired aortic valvular stenosis and insufficiency.

pected abnormalities of the aortic valvular and paravalvular regions (Fig. 9, *A–F*).

Utilizing the Seldinger technique, either a 14 or 15 gauge thin walled teflon catheter or a 7F or 8F radiopaque teflon catheter is threaded through the femoral artery into the ascending aorta. The spring wire tip of the small Seldinger wire is allowed to extend approximately 2 cm. beyond the catheter tip. It is a simple matter to manipulate the catheter through a normal aortic valve. In children, it is frequently possible to manipulate the catheter tip through the stenotic aortic valve into the left ventricle.

In calcific aortic stenosis, retrograde left ventricular catheterization is more difficult. If the ventricle is catheterized, cinecardioangiography is performed in the right anterior oblique and left anterior oblique projections. The catheter is then withdrawn into the ascending aorta and a third cineangiogram is made. If the left ventricle cannot be catheterized by this approach, an injection is made above the aortic valve and the patient is roentgenographed in the left anterior oblique position. The mobility of the valve, the degree of stenosis and the quantitation of insufficiency can be evaluated. The coronary arteries can also be evaluated. In such cases it is clinically desirable to obtain the pressure gradient across the left ventricular outflow tract by means of left ventricular catheterization by the transbronchial, transseptal or percutaneous apical approach.[23] The latter two methods are desirable because a catheter of sufficient caliber can be utilized for left ventricular angiography.

SUMMARY

1. The roentgenographic manifestations of 89 cases of proven aortic valvular and paravalvular disease were studied.

2. In aortic stenosis, the heart was frequently not enlarged. There was no correlation between cardiac size and the severity of the stenotic lesion.

3. Concentric hypertrophy of the left ventricle was noted in the vast majority of patients with aortic stenosis. This is roentgenographically manifest as a convex bulging of the lower one-third of the left cardiac border.

4. Dilatation of the left ventricle was usually noted in aortic insufficiency. The apex of the heart was displaced downward and posterior in the posteroanterior and left anterior oblique views.

5. Left atrial enlargement was common in patients with aortic valvular and subvalvular disease. This did not necessarily represent associated involvement of the mitral valve.

6. Dilatation of the ascending aorta was a common finding in congenital and acquired aortic valvular stenosis. It was equally as common in subvalvular aortic stenosis except that it was not present in the idiopathic hypertrophic type. It was absent in supravalvular stenosis.

7. There was no correlation of the degree of dilatation of the ascending aorta with the severity of the stenotic lesion.

8. The ascending aorta was rarely significantly dilated in aortic valvular insufficiency. If moderate to marked dilatation is present in a patient with aortic insufficiency, one should suspect significant associated aortic stenosis unless luetic aortitis or Marfan's syndrome is present.

9. In aortic stenosis increased pulsation was limited to the ascending aorta while in aortic insufficiency the entire aortic arch and descending aorta were hyperactive.

10. Calcification of the aortic valve was present in 85 per cent of patients with acquired aortic stenosis. It was rare in patients under 20 years of age.

11. Left heart catheterization and cinecardioangiography is the most accurate method by which aortic stenotic and regurgitant lesions may be studied.

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INTRACARDIAC MYXOMA DEMONSTRATED ON A VASCULAR SCAN*

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THE technique of vascular scanning utilizing radioactive materials was first described by Rejali *et al.*[4] in 1958. Vascular scanning was used at this time to evaluate cardiac size in patients with pericardial effusion. This technique was later modified by Bonte *et al.*[1] in 1961, and its use in evaluating para-aortic masses was pointed out. We modified this technique to some degree and reported additional cases of its use.[3] It is the purpose of this paper to describe a case in which the cardiac or vascular scan proved to be of benefit in demonstrating an intracardiac myxoma. To our knowledge this is the first such case recorded. The lesion was demonstrated using a vascular scanning technique. This case is to be reported in further detail and from a medical aspect elsewhere.[2]

REPORT OF A CASE

E. C., F54369. A 60 year old white married female was admitted to Duke Hospital with a history of congestive heart failure for the past 4 years. Two years prior to admission she developed complete heart block and one and one-half years prior to admission she began to have episodes of unconsciousness. Her first admission to the hospital was in August, 1961 for evaluation of the use of a permanent cardiac pacemaker. She continued to have syncopal episodes which became more frequent, and she was re-admitted on September 14, 1961.

The blood pressure was 100/60 and pulse 60 and irregular. There was a right ventricular heave without gross cardiomegaly on physical examination. The rhythm was grossly irregular and there was an easily heard mid-diastolic rumbling murmur. Chest roentgenograms (Fig. 1, *A* and *B*) revealed cardiomegaly and an electrocardiogram demonstrated complete heart block.

On September 20, 1961 a precordial scan (Fig. 2) was done using 300 μc of radioactive iodinated serum albumin. There was good visualization of the portion of the heart behind and to the right of the sternum; however, the portion of the heart to the left of the sternum showed a rather large defect appearing somewhat rounded and measuring about 8 cm. in diameter. The ascending aorta visualized well and appeared to be normal. This filling defect was believed to represent an intracardiac tumor, probably within the right ventricle. The possibility of a tumor or cyst of the pericardium displacing the right ventricle posteriorly could not be excluded.

The next day cardiac catheter studies and cinefluorograms revealed a large filling defect in the region of the right ventricle. This was felt to represent a large myxoma of the right ventricle.

Surgery revealed a large 6×6×5 cm. rounded tumor attached to the wall of a markedly enlarged right atrium in the region of the atrioventricular node. This was removed and the patient was returned from the operating room in good condition. She had an uneventful convalescence although she maintained her complete heart block.

DISCUSSION

This case represents the first intracardiac myxoma to be visualized on a vascular scan, so far as we have been able to ascertain. The technique is not new but is not widely used. It is a simple procedure and can be carried out with most routine scanning equipment. The test procedure itself causes no significant danger other than possibly that due to the total body irradiation delivered by injected iodine labeled albumin.

The use of human serum albumin is desirable as it remains in the blood over a long period of time and therefore permits an adequate scan of vascular structures; however, it also delivers more radiation to the patient than some of the more rapidly

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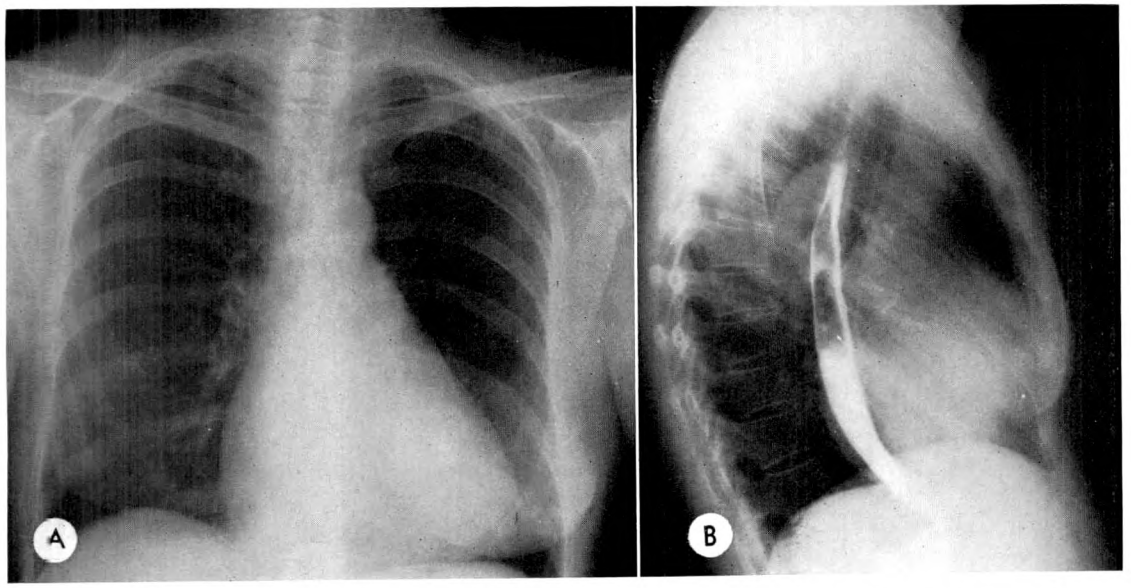


Fig. 1. Posteroanterior (*A*) and lateral (*B*) chest roentgenograms show generalized enlargement, but no specific heart chamber enlargement.

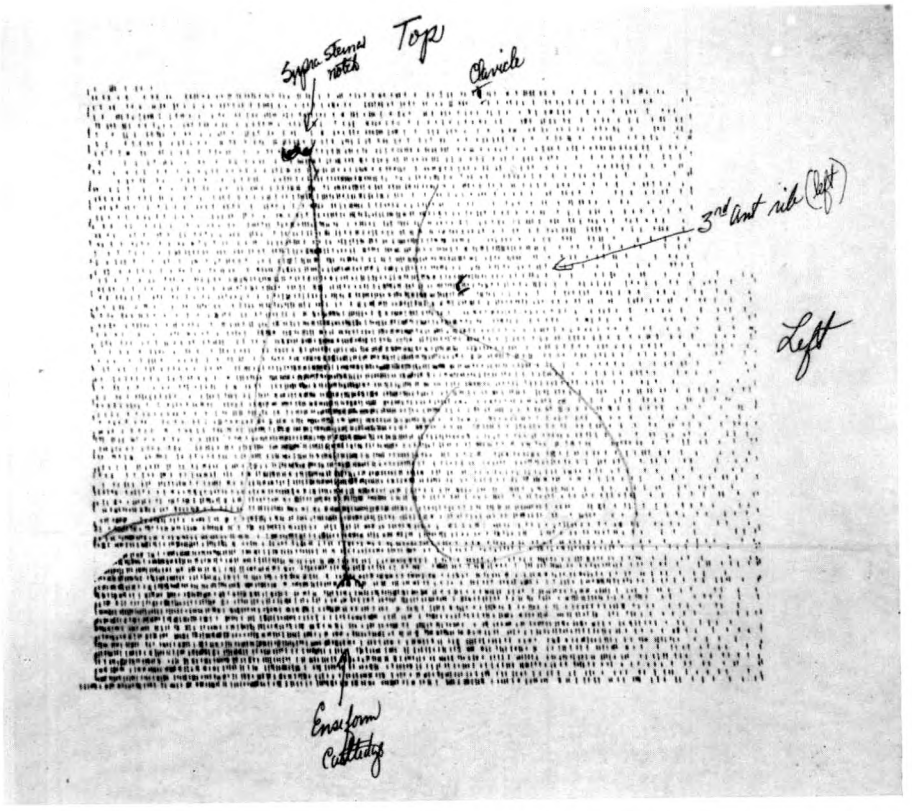


Fig. 2. Scanogram. The retrosternal portion of the heart and that to the right of the sternum are well visualized. The aorta appears normal. There is a rounded filling defect to the left of the sternum, representing a tumor of the right ventricular region.

excreted isotopes. The iodine is gradually separated from the albumin in the blood and much of the radioactive iodine is taken into the thyroid. For this reason most of our patients receive a thyroid blocking dose of iodine prior to injection of the isotope.

The interpretation of the scan is best carried out by its superimposition on a roentgenogram taken in a similar position. One must consider, however, that there may be slight changes due to projection. The highly vascular areas as well as those areas with decreased vascularity may be delineated. It must be remembered, in rendering an interpretation of such a scan, that a displacement of vascular structure away from the chest wall, *i.e.*, increasing the distance from the scintillation crystal to the radioactivity, may give a picture similar to that of decreased radioactivity over an area. It was for this reason that the possibility of a pericardial cyst or tumor displacing the blood volume posteriorly could not be excluded.

It was realized that the left ventricle was not the most probable location for a tumor, but the appearance of the scan left no alternative. The marked enlargement of the right atrium due to the prolonged partial obstruction of the tricuspid valve placed the lesion, as well as the right atrium itself, into the area commonly designated on the posteroanterior projection as ventricular.

SUMMARY

A vascular scan is described in which an intracardiac filling defect, proving to be an intra-atrial myxoma, was demonstrated. The technique as well as the differential diagnosis based on a vascular scan is reported.

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Vol. 88, No. 1

ANOMALIES (PSEUDOCOARCTATION) OF THE ARCH OF THE AORTA*

REPORT OF 8 NEW AND REVIEW OF 8 PREVIOUSLY PUBLISHED CASES

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ANOMALIES (pseudocoarctation) of the arch of the aorta are uncommon; only about 40 cases have been previously reported. Their recognition, however, is important because they may be mistaken for true coarctation, aneurysm, and/or mediastinal tumor. Conventional roentgenography, roentgenoscopy, and angiocardiography provide considerable differential diagnostic data, but the definitive diagnosis depends upon the demonstration of unimpeded blood flow into the descending aorta, abdomen, and legs.

The purpose of this paper is to establish criteria for the diagnosis of pseudocoarctation of the arch of the aorta by relating the experience in a series of 16 patients at The New York Hospital—Cornell Medical Center, 8 of whom are newly reported. Because such anomalies of the aortic arch may simulate true coarctation, and because embryologically the two conditions appear to be closely related, the term "pseudocoarctation," as originally proposed at this center, is preferred to "kinking" or "buckling" of the aortic arch.

REPORT OF CASES

Table 1 presents the pertinent clinical and roentgen findings for all the cases reported.

CASE 1. A fifty-seven year old bartender (N.Y.H. No. 742277) was admitted on July 4, 1956, because of pain in the left leg of ten weeks' duration. This began in the left buttock with radiation down to the superior aspect of the thigh, the anterior portion of the leg, and the dorsum of the foot.

Physical examination showed a spastic gait, wasting, weakness and clonus of the left leg. There was also atrophy of the left buttock, diminished vibratory sensations in the great toes, and diminished pin-prick sensations bilaterally below the level of the fifth thoracic vertebra. The cardiovascular system was normal. The blood pressure readings were as follows: right arm, 110/62; left arm, 107/54; right leg, 132/88; and left leg, 158/88 mm. Hg, respectively.

The frontal roentgenogram of the chest (Fig. 1*A*) showed a long heart with overdistended voluminous lungs. The ascending aorta appeared dilated. The left upper cardiac silhouette, beginning at the apex of the lung, contained three distinct outwardly convex structures, which were also recognized in the frontal esophagram (Fig. 1*B*). The left anterior oblique view with the esophagus outlined with barium (Fig. 1*C*), indicated that the lower structure was in reality the dilated descending aorta. Roentgenoscopic study of the left upper mediastinal structures showed aortic type pulsations, and, because of these, angiocardiography was recommended. Prior to this, and because a mediastinal tumor with nerve root compressions was suspected, a left scalene node biopsy was done; this proved to be negative for tumor. Angiocardiography revealed a mildly dilated ascending aorta which extended high in the neck. The transverse aorta was tortuous and there was dilatation of the left subclavian artery. Three centimeters beyond the origin of the left subclavian artery, the arch of the aorta was kinked and joined a curved, dilated and poststenotic appearing descending aorta (Fig. 1*D*).

Since the left superior mediastinal masses were shown to be vascular, a search was made to explain the patient's symptoms and signs. A myelographic examination showed cervical hypertrophic osteoarthritis with ridging of the foramen and a herniated intervertebral disk between the bodies of the fourth and fifth lumbar vertebrae.

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TABLE I

CLINICAL AND ROENTGEN FINDINGS IN PSEUDOCOARCTATION OF THE ARCH OF THE AORTA

Case No.	Age	Sex	Symptoms	Associated Disease	Cardiac Murmurs	ECG	Blood Pressure (mm. Hg)
I	57	M	Pain in legs	Cervical osteoarthritis, herniated lumbar disk, pulmonary emphysema	None	Normal	R.A. = 110/62 R.L. = 132/88 L.A. = 107/54 L.L. = 158/88
II	58	F	None	Hypertension, small trichoepithelioma of face	Loud, low-pitched (Grade 3) systolic over base of heart transmitted to neck with systolic at apex (Grade 2)	Left axis deviation	R.A. = 172/88 R.L. = 200/120 L.A. = 176/86 L.L. = 180/120
III	53	M	None	Late latent syphilis	Systolic (Grade 1) at base	Normal	130/70
IV	30	M	None	None	Systolic at base, rough systolic over entire precordium, loudest at third interspace and sternum	Normal	R.A. = 150/90 L.A. = 130/70
V	32	F	None	None	Systolic at base and left of sternum	Normal	R.A. = 124/70 R.L. = 200/160
VI	41	F	Joint pains	None	None	Normal	130/90
VII	55	M	None	None	None	Normal	R.A. = 112/78
VIII	22	F	None	Corrected transposition with dextrorotation of heart	Short blowing basal and apical systolic	Shift to right with transition zone between V_1 and V_2 semivertical position	R.A. = 114/76 R.L. = 150/90 L.A. = 116/72 L.L. = 150/90
IX[9]	49	M	None		Systolic at aortic area		R.A. = 140/85 R.L. = 150/90 L.A. = 158/105 L.A. = 158/105 L.L. = 152/108
X[9]	37	F	None	Subacute bacterial endocarditis	None	Normal	
XI[10]	44	M	None	Hypertension	None		R.A. = 170/100
XII[18]	26	F	None	Aneurysm of sinuses of Valsalva	Systolic (Grade 3) over entire base, loudest along left sternal border and transmitted to neck and axilla	Normal	R.A. = 126/90 R.L. = 140/120 L.A. = 130/90 L.L. = 132/110
XIII[21]	12	M	None	Aortic stenosis, bicuspid aortic valve (found at operation)	Systolic (Grade 4) at base radiating to neck, axilla and precordial systolic thrill into neck	Left ventricular hypertrophy	R.A. = 94/70 L.L. = 90/74
XIV[21]	14	F	None	Aortic stenosis	Harsh systolic (Grade 3–5) over entire precordium, loudest along left sternal border with systolic thrill at base transmitted into neck	Normal	R.A. = 110/75 R.L. = 105/70 L.A. = 100/75 L.L. = 105/70
XV[21]	10	M	None	Aortic stenosis	Systolic (Grade 5) at base radiating into neck and back with systolic thrill at base extending into neck	Left axis deviation	R.A. = 100/60 R.A. = 104/71 (direct reading)
XVI[22]	42	M	Chronic heart failure	Mild aortic stenosis, bicuspid aortic valve, endocardial fibroelastosis (found at postmortem)	Systolic along left sternal border, also third heart sound (gallop)	Left axis deviation and left ventricular hypertrophy	R.A. = 94/80 L.L. = 104/80

R.A. = right arm. R.L. = right leg. L.A. = left arm. L.L. = left leg.

On July 27, 1956, a herniated disk of the fourth lumbar vertebra was removed and recovery was uneventful. The left leg pain was relieved, but clonus persisted. Accordingly, two weeks later, cervical laminectomy and bilateral section of the dentate ligaments at the level of the fourth to sixth cervical vertebrae were performed. Following these procedures the patient improved, but he continued to have pains in the legs and thighs. He was last seen in the clinic on December 4, 1958.

CASE II. A fifty-eight year old woman (N.Y.H. No. 818934) was admitted on March 19, 1959, because of a small (1 × 1 cm.) induration of the right cheek, which was excised. On histologic

Table I—(*Continued*)

Conventional Roentgenography	Aorta	Angiocardiography	Heart	Remarks and Follow-up
Indented left superior mediastinal masses (Fig. 1*A*), reversed figure 3 in esophagram (Fig. 1*B*), poststenotic dilatation of descending aorta (Fig 1*C*).	High aortic arch with prominent poststenotic dilatation of descending aorta (Fig. 1*D*)		Normal	Improved after laminectomy but required physiotherapy for leg pains when last seen in 1958
Large aortic knob lined with calcium (Fig. 2*A*), calcifications along posterior wall of poststenotically dilated descending aorta (Fig. 2*B*).	Calcium-lined wall of poststenotically dilated portion of descending aorta (Fig. 2, *C* and *D*)		Normal	Asymptomatic, facial trichoepithelioma excised
Unduly prominent aortic knob (Fig. 3*A*)	Poststenotic dilatation of descending aorta with linear calcium in anterior wall (Fig. 3*B*)		Normal	Although seropositive, syphilis remains inactive forty years after chancre
Double aortic knob (Fig. 4)			Normal	Remains asymptomatic
Calcium-lined mass (3×5 cm.) in left superior mediastinum (Fig. 5, *A* and *B*)	Calcium-lined posterior wall of poststenotic descending aorta (Fig. 5, *C* and *D*)		Normal	Has had 2 later pregnancies and continues to be asymptomatic
Pulsatile mass, left superior mediastinum	High aortic arch with poststenotic dilatation of descending aorta (Fig. 6)		Normal	Etiology of joint pains not determined, patient otherwise well
Unusually prominent aortic knob (Fig. 7, *A* and *B*)	Unusually wide aortic arch with a large buckle at top of descending aorta (Fig. 7, *C* and *D*)		Normal	Developed fainting spells, etiology not determined
Dextrorotation of heart with aorta descending along left cardiac silhouette (Fig. 8*A*)	Pseudocoarctation (Fig. 8*B*)		Classic corrected transposition with dextrorotation of heart (Fig. 8, *B–D*)	Hospital employee, dextrocardia demonstrated on routine roentgenogram of chest
Double rounded shadows in left superior mediastinum; left ventricle enlarged (Fig. 9*A*)	Unusually dilated left subclavian artery (Fig. 9, *B* and *C*)		Normal	No follow-up
Widened arch of aorta	Dilated ascending aorta with unfolding of descending aorta (Fig. 10, *A–D*)		Normal	Developed bacterial endocarditis (alpha streptococcus) seven years after these studies, treated with antibiotics, negative cultures, developed pulmonary edema and died one year later; at autopsy: healed perforated aortic miliary abscesses of myocardium, cusp, cardiac dilatation and hypertrophy, mural thrombi in both atria
Widened mediastinal shadow adjacent to aortic knob, displacement of esophagus (Fig. 11, *A* and *B*)	Constriction of aortic arch and poststenotic dilatation of descending aorta (Fig. 11, *C*; and Fig. 11, *D* and *E*)		Slight enlargement of left ventricular wall	No follow-up
Left superior mediastinal mass forming double aortic shadow, poststenotically dilated descending aorta (Fig. 12, *A–C*)	Classic appearance of true coarctation except for absent collateral vessels (Fig. 12, *D–G*)		Normal	Remains asymptomatic, recently delivered of child, no complications
Dilatation of ascending aorta, enlarged left ventricle	Deformed aortic valve, marked poststenotic dilatation of ascending aorta, aortic kink, and poststenotic dilatation of descending aorta but with absent collaterals		Enlarged left ventricle	Aortic valvulotomy with open-heart surgery, good result, three year follow-up
Widening of descending aorta with double contour in left superior mediastinum and large left ventricle	Dilated ascending aorta, deformed aortic arch, and poststenotic dilatation of descending aorta without collaterals		Large left ventricular wall	No follow-up past two years
Dilated ascending aorta, prominence of aortic knob, and left ventricular enlargement	Aortic stenosis and poststenotic widening of ascending aorta		Left ventricular enlargement	Aortic valvulotomy planned
Marked cardiac enlargement with dilatation of ascending aorta				Died of heart failure, autopsy revealed bicuspid aortic valve, parietal endocardial fibrosis, fibroelastosis, dilatation of both atria, dilatation and hypertrophy of both ventricles

study it was found to be a trichoepithelioma. The blood pressure readings were 172/88 and 200/120 mm. Hg in the right arm and right leg, respectively.

Routine roentgenography of the chest (Fig. 2*A*) showed a prominent translucent calcium-lined shadow in the position of the aortic knob. The left anterior oblique roentgenogram of the chest demonstrated that the linear calcifications were in the posterior wall of the dilated descending aorta (Fig. 2*B*). Angiocardiography showed kinking of the arch of the aorta 2.5 cm. beyond the origin of the left subclavian artery with poststenotic dilatation of the descending aorta, which contained calcification in the posterior wall (Fig. 2, *C* and *D*).

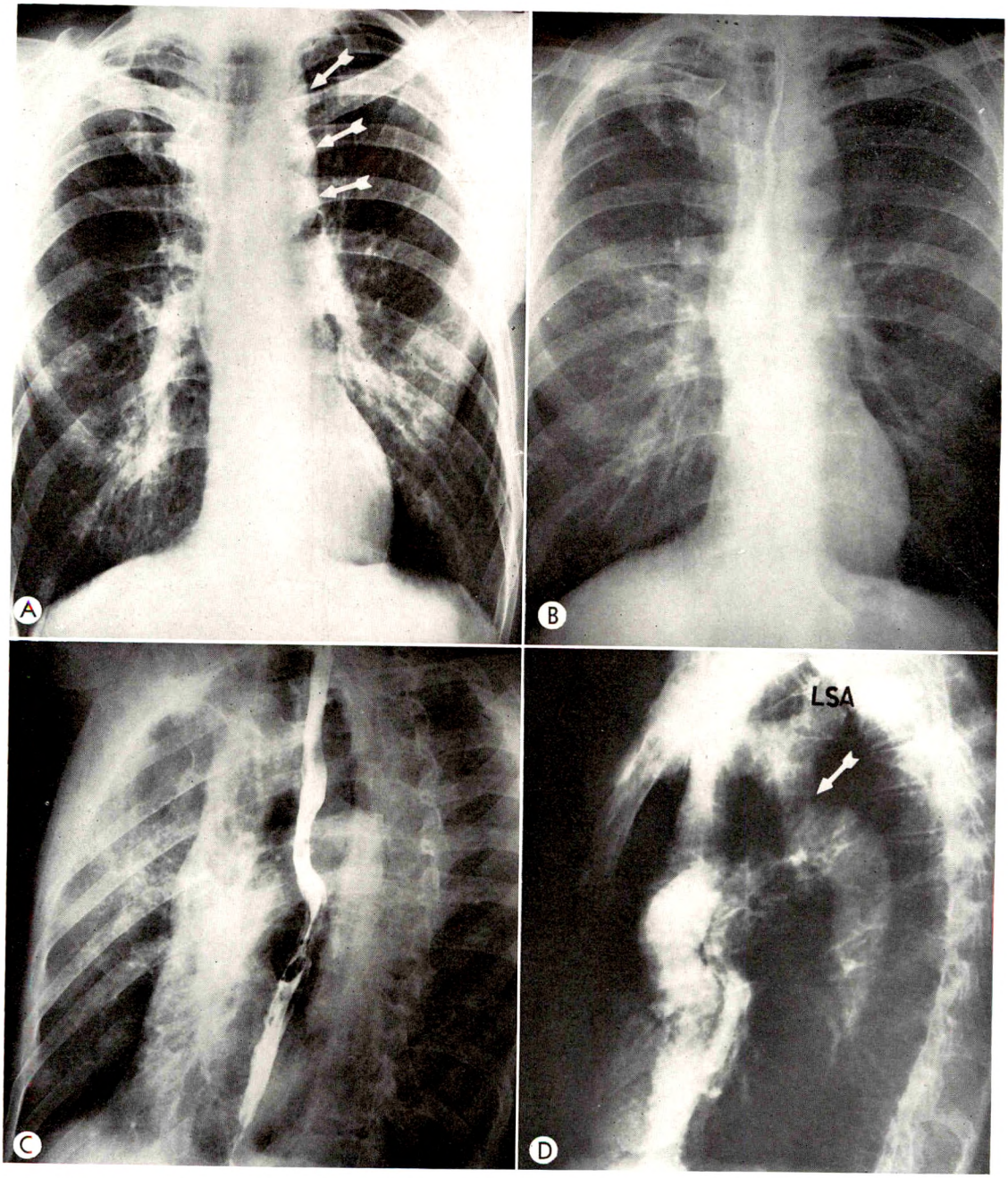


FIG. 1. Case 1. Pseudocoarctation of the aorta in a fifty-seven year old man with neurologic complaints owing to spinal canal pressure from cervical osteoarthritis and a herniated disk of the lumbar spine. (*A*) Conventional frontal teleroentgenogram of the chest showing a ptotic, normal-sized heart with mild dilatation of the ascending aorta. There are three convex shadows (arrows) along the left superior mediastinal border of the heart. (*B*) Frontal esophagram demonstrating indentation of the esophagus by two of the lower vascular masses. (*C*) Left anterior oblique esophagram revealing that indentation of the upper part of the esophagus is caused by the aortic arch; below is the characteristic displacement of the esophagus by the dilated poststenotic descending aorta. Note the mild dilatation of the ascending aorta. (*D*) Left anterior oblique angiocardiogram showing a mildly dilated ascending aorta which proceeds directly upward. The transverse aorta is short and is joined at the arch by a dilated left subclavian artery (LSA), which is responsible for the upper left superior mediastinal mass. A short stub of the descending aorta (aortic knob) forms the middle mass. Below, there is a point of narrowing (arrow), usually seen at the site of coarctation. Distally, there is poststenotic dilatation and kinking of the descending aorta. This is responsible for the lowermost deformity of the esophagus.

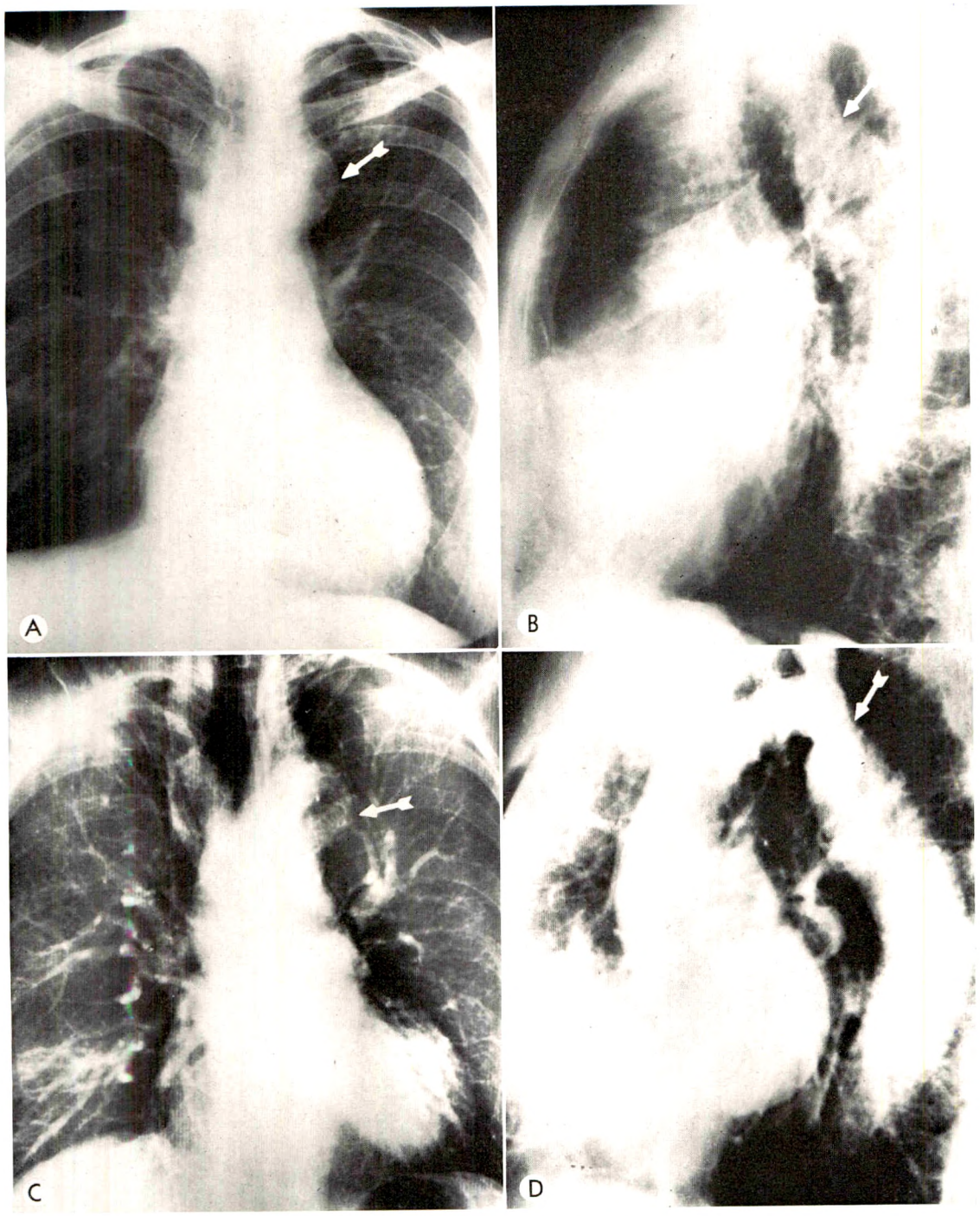


FIG. 2. Case II. Pseudocoarctation of the arch of the aorta in an asymptomatic fifty-eight year old woman with hypertension. (*A*) Conventional frontal teleroentgenogram of the chest showing some enlargement of the left ventricle. The shadow in the region of the aortic knob (arrow) is translucent and lined by calcium. (*B*) Conventional left anterior oblique view of the chest showing kinking of the arch of the aorta (arrow). Note the calcium-lined posterior wall of the poststenotically dilated descending aorta. (*C*) Frontal angiocardiogram showing enlargement of the left ventricular wall (owing to hypertension) and deformity of the arch of the aorta. The transverse aorta is of normal caliber, but the arch of the aorta is dilated and appears translucent because of encroachment into the lung field; it is lined with calcium (arrow). There are no collateral arteries. (*D*) Left anterior oblique angiocardiogram revealing the detailed anatomy of the aortic congenital anomaly. Just beyond the origin of the left subclavian artery (arrow), the descending aorta becomes dilated and tortuous. Again note the calcium-lined posterior wall of the poststenotic segment of the descending aorta. Collateral arteries are not present.

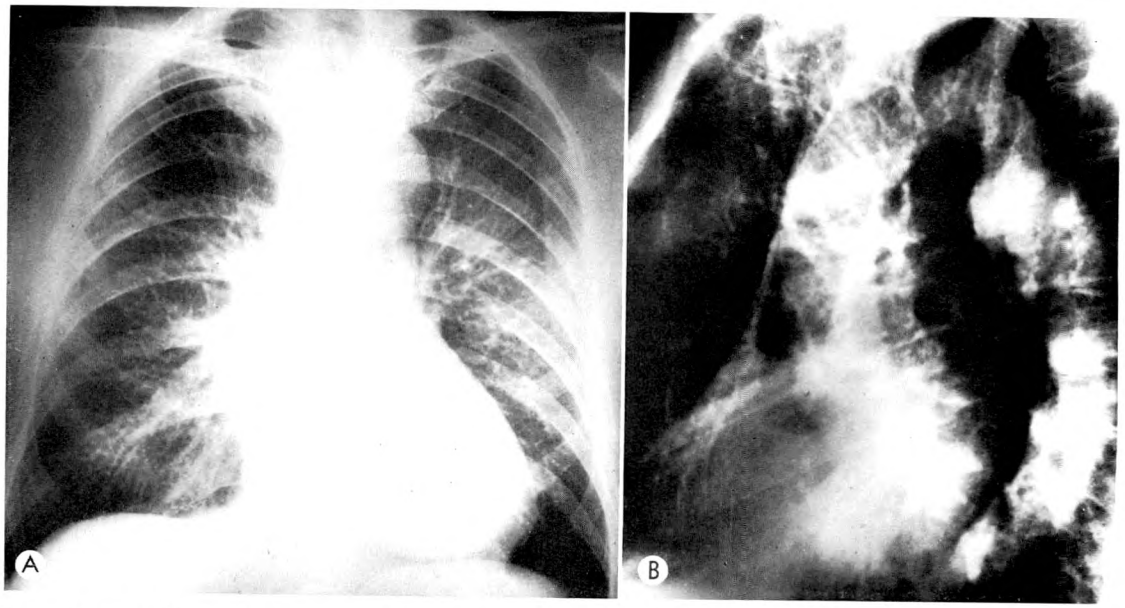


FIG. 3. Case III. Pseudocoarctation of the aorta mistaken for an aneurysm in a fifty-three year old seropositive syphilitic man. (*A*) Frontal teleroentgenogram of the chest showing a large, prominent, translucent shadow in the region of the aortic knob. (*B*) Left anterior oblique angiocardiogram revealing the classic features of pseudocoarctation, *i.e.*, kinking of the descending aorta with poststenotic dilatation of the descending aorta, but without collateral arteries.

CASE III. A fifty-three year old asymptomatic salesman (N.Y.H. No. 470357) was referred for angiocardiography after a routine roentgenogram of the chest showed undue dilatation of the arch of the aorta. In 1918, while in France with the armed service, he had had a chancre and was treated with salversan for two months. He was discharged from the army at the end of the war. In 1920, a routine serologic test had been positive and he had been treated with antisyphilitic drugs for a period of sixteen months. Thirty years later, another serologic test was positive, and because of this he was referred to the syphilitic clinic of this hospital.

On examination the patient was found to be well developed and well nourished. He weighed 61.6 kg. and measured 166 cm. A soft systolic (Grade 1) murmur was heard at the base of the heart and extended into the neck. The heart was normal in size, the rate was regular, and the groin and leg pulses were palpable. Neurologic examination was within the limits of normal. Blood serologic tests (Kline, Kahn, and Mazzini) were positive, but the spinal fluid tests (Kahn and Kline) were negative. The patient was classified as a late latent seroresistant syphilitic who required no antisyphilitic treatment.

In 1953, as part of a routine follow-up, a roentgenogram of the chest was secured and revealed undue prominence of the aortic knob (Fig. 3*A*). Angiocardiography done on December 1, 1953 disclosed a normal-sized left ventricle and ascending aorta; 3 cm. beyond the left subclavian artery the aortic arch became kinked and the descending aorta was dilated and contained calcium in the upper anterior wall (Fig. 3*B*). On the latest visit, August 8, 1958, the patient was still asymptomatic and the physical examination was within the limits of normal.

CASE IV. An asymptomatic thirty year old man (reported through the courtesy of Dr. William D. Stubenbord) had had a heart murmur which prevented him from entering the armed services. At another hospital, coarctation of the aorta was diagnosed and an operation had been advised. On physical examination he was found to be well developed and well nourished and weighed 139 pounds. The pulse had a regular sinus rhythm, with a rate of 90 per minute. The heart was not enlarged and the sounds were of good quality. The pulmonic second sound was louder than the aortic second sound. A rough systolic murmur was heard all over the precordium, loudest at the sternum in the third left interspace. The blood pressure readings were: right arm, 150/90; left arm, 130/70 mm. Hg. The femoral and dorsalis pedis

pulses were palpable. No collateral arteries were palpable in the neck or thorax. The roentgenogram of the chest (Fig. 4) showed the classic double shadow of the left superior mediastinum with translucency of the upper mass. Rib notching was not seen, and the heart was not enlarged. Angiocardiography was recommended but refused by the patient.

Case v. A thirty-two year old asymptomatic housewife (N.Y.H. No. 667231) was referred after angiocardiography at another hospital had disclosed "an aneurysm of the aorta," for which an operation had been advised. Prior to becoming pregnant, the patient had had a routine physical examination and, because a cardiac murmur was heard, a roentgenogram of the chest was ordered. Following this, angiocardiography was performed.

The patient was well developed and well nourished; weight was 150 pounds, and height, 5 feet $7\frac{1}{2}$ inches. A systolic murmur was heard at the base of the heart just to the left of the sternum. The blood pressure in the right arm was 124/70 mm. Hg; in the right leg it was 200/160. The electrocardiogram was normal. Conventional roentgenograms of the chest showed a dense lobulated mass in the left upper mediastinum which contained calcium along its posterior wall (Fig. 5, *A* and *B*). Angiocardiography on September 29, 1953, showed a vascular shadow occupying the left apex of the lung area; in the left anterior oblique view it was seen to represent an enlarged arch of the aorta and a mildly dilated poststenotic tortuous descending aorta (Fig. 5, *C* and *D*). Calcification lining the posterior wall of the poststenotic descending aorta was evident. When last seen in 1956, the patient was asymptomatic and had had two pregnancies without ill effect.

Case vi. A forty-one year old widow (N.Y.H. No. 655903) was admitted to the outpatient clinic with complaints of numbness and swelling of the hands, and low back pain. Physical examination was normal except for obesity; she weighed 87.5 kg. and measured 171 cm. The heart was normal in size and there were no murmurs; the blood pressure was 130/90 mm. Hg. The pulses in the groins and legs were normal.

Roentgenograms of the hands did not show osseous deformity. A roentgenogram of the chest showed the heart to be normal in size, but there was a pulsatile mass in the left superior mediastinum. Angiocardiography was performed on July 28, 1953. It disclosed an unusually elongated arch of the aorta which reached the level of the body of the second thoracic vertebra. It measured 20 mm. in diameter at a point just beyond the origin of the left subclavian artery. Below, at the upper level of the body of the fourth thoracic vertebra, the aorta was dilated (32 mm.) (Fig. 6). The etiology of the joint pains was not determined.

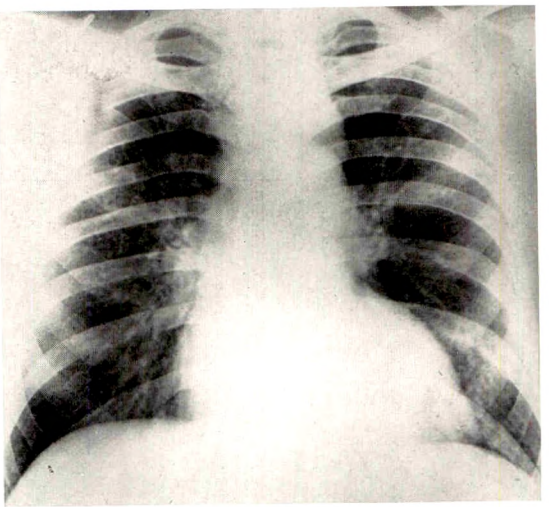


Fig. 4. Case iv. Pseudocoarctation of the aorta in a thirty year old man mistakenly diagnosed as coarctation. Frontal teleroentgenogram shows the classic double masses along the left superior mediastinum.

Case vii. A fifty-five year old asymptomatic salesman (N.Y.H. No. 133354) was referred because of an unusually prominent aortic knob. He weighed 172 pounds and was 6 feet 2 inches in height. The blood pressure in the right arm was 112/78 and the pulses in the groins, legs and feet were normal. The electrocardiogram was also normal. Conventional roentgenograms of the chest showed the heart to be normal in size. The aortic knob was unusually prominent and extended well into the left hemithorax (Fig. 7, *A* and *B*). Angiocardiography, done on October 2, 1951, showed a very wide aortic arch with kinking just beyond the origin of the left subclavian artery and tortuosity of the descending aorta (Fig. 7, *C* and *D*). When last examined in 1957, he had been well except for syncope, the etiology of which could not be determined.

Case viii. An asymptomatic twenty-two year old woman (N.Y.H. No. 568471) had had a routine pre-employment roentgenogram of the chest which disclosed dextrocardia. She had first learned that her heart was in the right

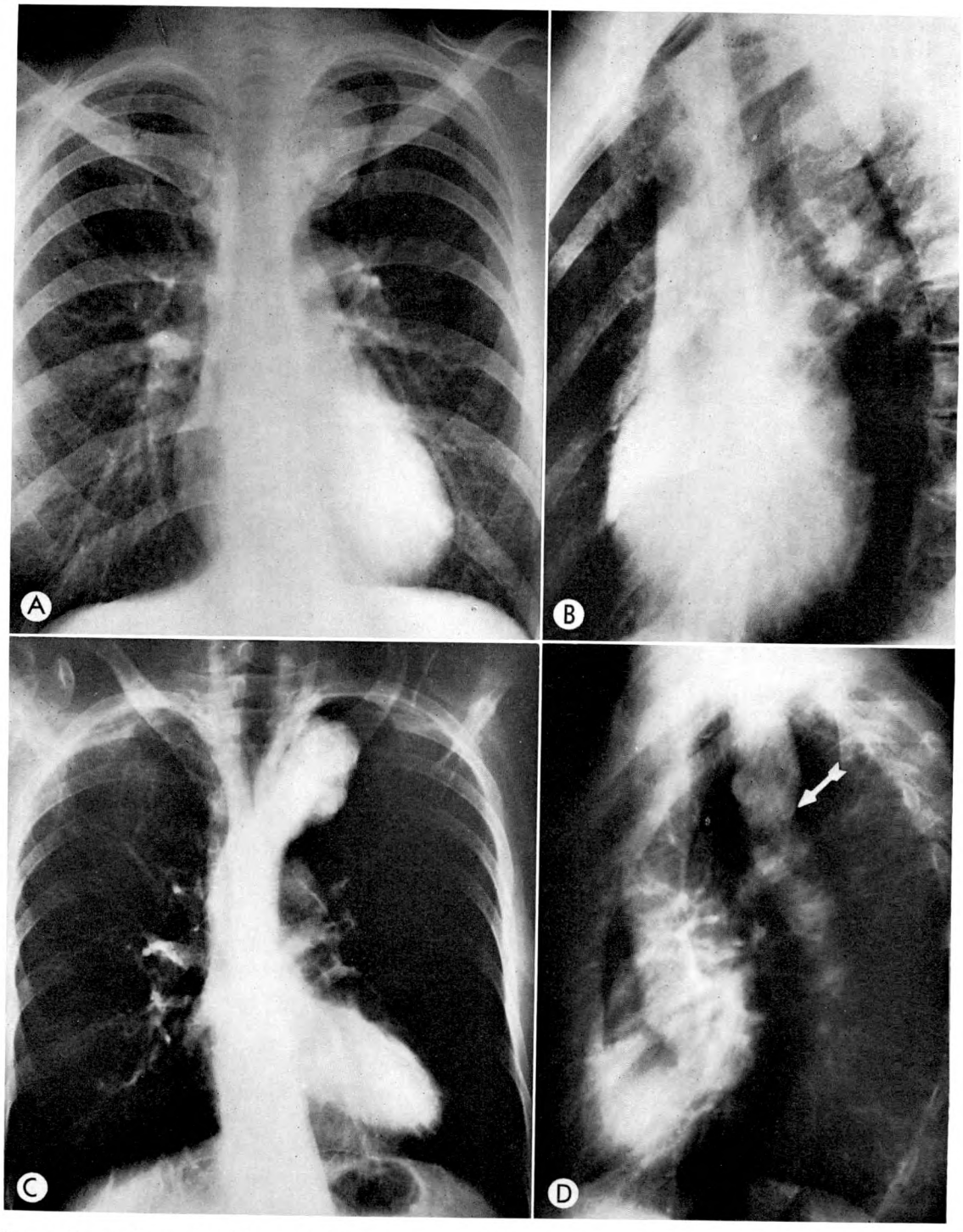


FIG. 5. Case v. Anomaly of the arch of the aorta (pseudocoarctation) mistakenly diagnosed as an aneurysm in a thirty-two year old woman. (*A*) Frontal teleroentgenogram of the chest showing a calcium-lined mass high in the left superior mediastinum. (*B*) Left anterior oblique view showing the unusually long aorta extending into the apex of the left lung. (*C*) Frontal teleangiocardiogram showing the normal caliber of the transverse aorta with the coiled, dilated and calcium-lined posterior wall of the arch of the aorta. (*D*) Left anterior oblique teleangiocardiogram showing the dilated, deformed, kinked, and tortuous descending aorta. One kink contains a plaque of calcium (arrow).

hemithorax when she was six years old, but she had always been without symptoms and had not required medical care. On physical examination she was found to be tall and thin, weighed 49.6 kg., and her height was 161 cm. The blood pressure readings were as follows: right arm, 114/76; right leg, 150/90; left arm, 116/72; left leg, 150/90 mm. Hg. The heart was displaced into the right hemithorax; the right border of cardiac dullness was in the right fifth interspace beyond the midclavicular line; the position of maximum impulse was not determined; a thrill was not present. A short, blowing, systolic murmur was heard at the base and apex of the heart. The pulses in the groins and legs were palpable, equal and normal.

The roentgenogram of the chest (Fig. 8*A*) showed rotation of the heart into the right hemithorax. The left cardiac silhouette, however, contained the aorta, and the left diaphragm was elevated. Gastrointestinal studies were normal. Angiocardiography on May 25, 1950, disclosed corrected transposition of the heart and great vessels, dextrorotation of the heart, and a deformed arch of the aorta which was kinked 2.5 cm. beyond the origin of the left subclavian artery; below, there was a classic poststenotic dilatation of the descending aorta, but arterial collaterals were not evident (Fig. 8, *B*, *C* and *D*).

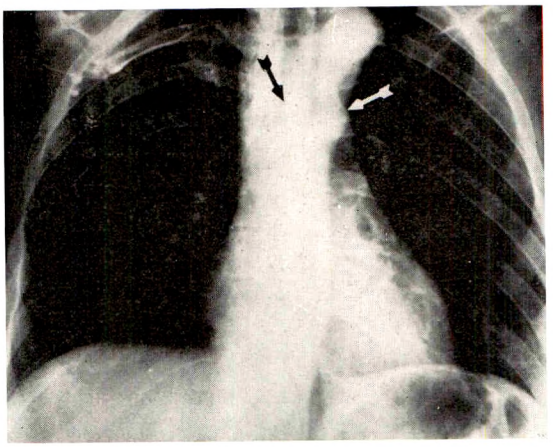


Fig. 6. Case VI. Pseudocoarctation of the arch of the aorta discovered at routine roentgenographic study of the chest in an asymptomatic woman of forty-one years. Frontal teleangiocardiogram showing an unusually high aortic knob and, below, a poststenotically dilated descending aorta (arrows).

DISCUSSION

CASES FROM THE LITERATURE

Anomalies of the arch of the aorta (pseudocoarctation) were described in 1951 by Dotter and Steinberg[9] who reported 2 patients with what we now call pseudocoarctation and commented on its resemblance to true coarctation. In the same year Robb[15] reported a case while Souders *et al.*[17] published data on 3 patients whose lesions were mistaken for mediastinal tumors, but who were classified as having subclinical coarctation. In 1952, Dotter and Steinberg [10] reported another case, and proposed that this deformity of the arch of the aorta be called pseudocoarctation. In 1955, Di Guglielmo and Guttadauro[8] described 2 patients with aortic arch deformities (pseudocoarctation), but they called the anomaly "kinking" of the aorta. In 1956, again from this center,[18] a patient with dilatation of the ascending aorta, aneurysmal dilatation of the aortic sinuses, and a deformity of the arch of the aorta (pseudocoarctation), but without aortic constriction, was described. Shortly thereafter Bruwer and Burchell,[5] Vaughan,[24] and Bruwer[6] also reported cases of anomalies of the arch of the aorta (pseudocoarctation) but they preferred to call them "kinking" of the aortic arch. In 1957 Samuel and Morris[16] reported 2 patients, and in 1958 Stevens,[23] in reporting 4 new cases of aortic arch anomalies, suggested that they be called "buckling" of the aortic arch. Pattinson and Grainger[13] in 1959 reported 5 additional cases of aortic arch anomalies under the title of "congenital kinking of the aortic arch." Two cases were also reported recently by Barrett and Verney,[1] who called special attention to the fusiform aneurysmal dilatation of the descending aorta. Gudbjerg and Petersen,[12] after visualizing the thoracic aorta and measuring the intra-arterial pressure proximal and distal to an apparent point of stenosis in 56 cases suspected of having coarctation of the thoracic aorta, found 4 patients who satisfied the criteria of psuedocoarctation;[18] *i.e.*, there was dilatation of the ascending aorta with apparent aortic constriction at the arch but without luminal occlusion (as determined by hemodynamic studies) or the develop-

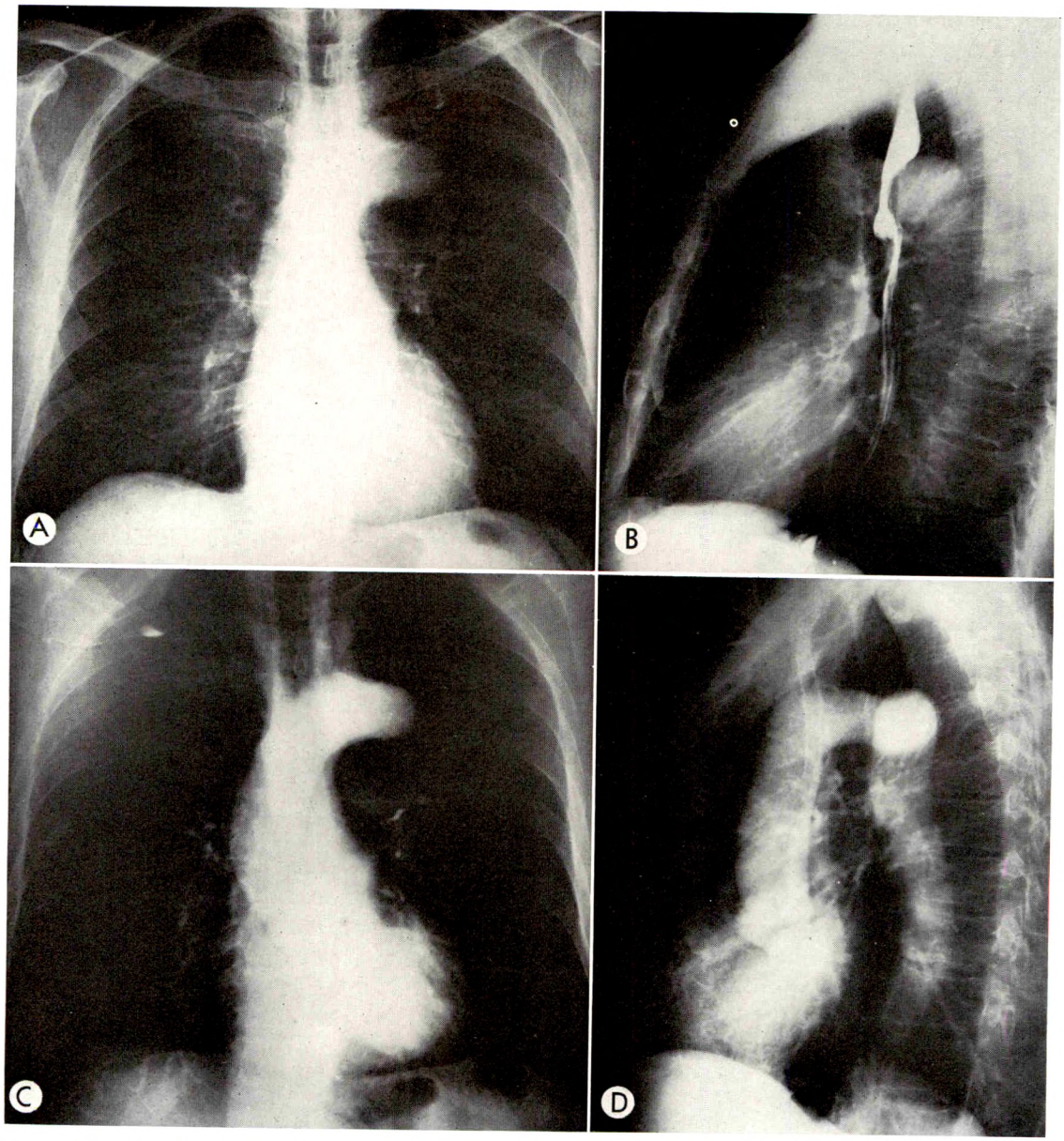


Fig. 7. Case VII. An abnormally prominent aortic knob in an asymptomatic fifty-five year old man. (*A*) Frontal teleroentgenogram of the chest showing an extremely laterally elongated aortic knob shadow. (*B*) Lateral esophagram showing anterior indentation of the wall of the esophagus by a dense aortic shadow. (*C*) Frontal teleangiocardiogram showing the normal left ventricle and very prominent aortic knob. (*D*) Left anterior oblique teleangiocardiogram disclosing the "end on" aspect of the arch of the aorta, apparently due to an unusually tortuous, widened and kinked arch and descending aorta.

ment of collateral arterial circulation. Finally, 4 new cases with congenital aortic stenosis (2 with bicuspid aortic valves) and pseudocoarctation were recently reported from this center.[22]

EMBRYOLOGY

Embryologically, pseudocoarctation seems to be closely related to true coarctation, the difference being that the aortic lumen is not compromised in pseudocoarctation. Pattinson and Grainger[13] have pointed out that Barry's[2] description of the complex changes that occur in the embryonic aortic arch during development may explain such aortic arch anomalies. In organogenesis

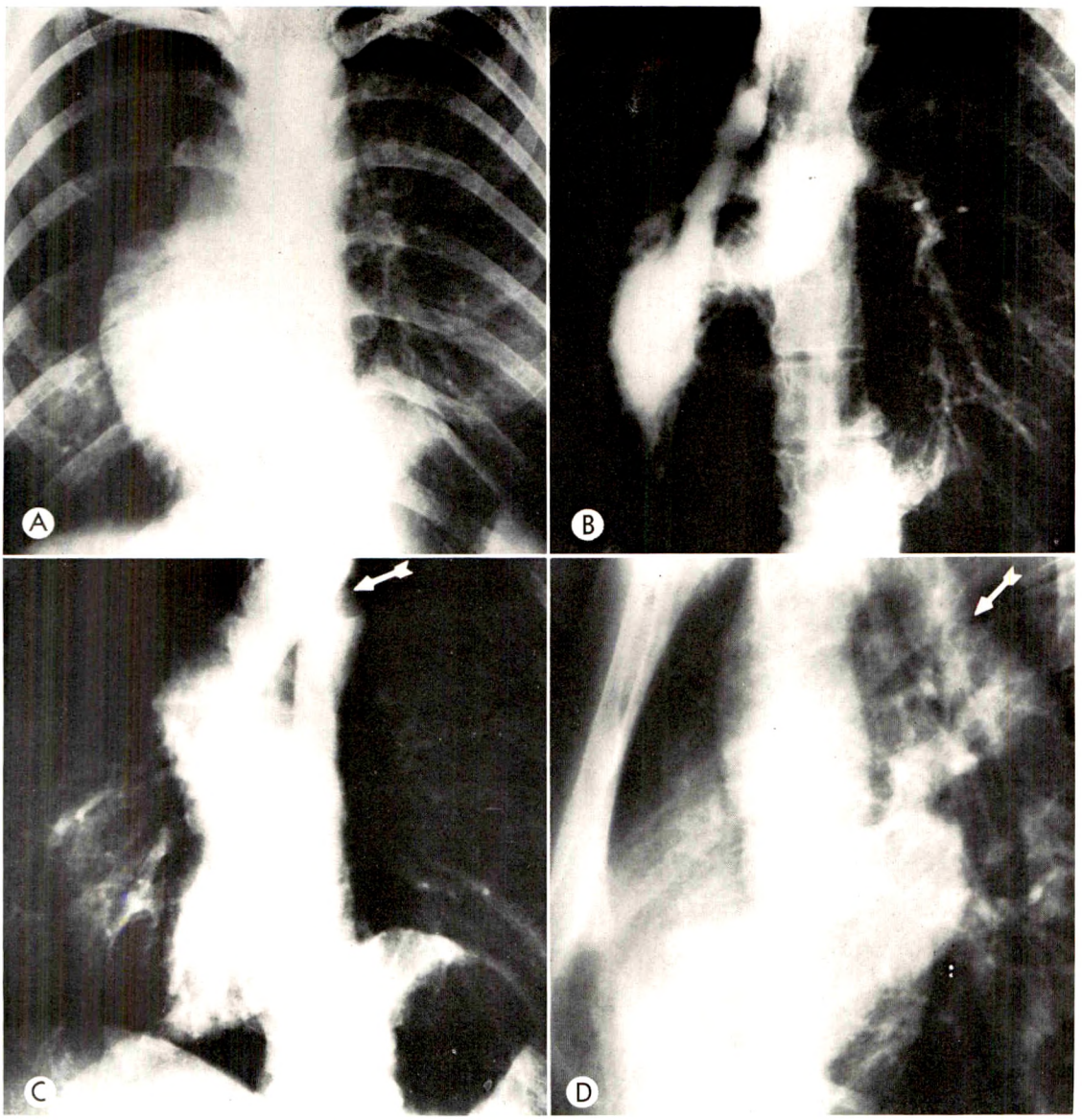


Fig. 8. Case VIII. Pseudocoarctation in an asymptomatic twenty-two year old woman with dextrorotation of the heart, corrected transposition of the great vessels, and eventration of the left diaphragm. (*A*) Frontal teleroentgenogram of the chest showing the heart displaced into the right hemithorax; the left diaphragm is elevated. (*B*) Frontal angiocardiogram showing rotation of the right atrium and ventricle into the right hemithorax; the pulmonary artery is centrally placed. (*C*) Frontal angiocardiogram showing dextrorotation of the left atrium, ventricle and aorta. The aorta contains a kink (arrow) 2 cm. beyond the origin of the left subclavian artery and it descends along the left cardiac silhouette in the manner characteristic of corrected transposition of the great vessels. (*D*) Left anterior oblique angiocardiogram showing kinking of the aorta below the left subclavian artery (arrow) and poststenotic dilatation of the descending aorta.

some arterial segments elongate while others become greatly contracted; one of these changes occurs in the seventh dorsal intersegmental arteries when they undergo a cephalad shift and become the origin of the subclavian arteries. When the heart descends into the thorax, the subclavian artery remains relatively far cephalad; there is associated compression of the third to seventh segments of the dorsal aortic roots and of the left fourth arch segments. Failure of compression of these segments during development results in an abnormally long aortic arch which twists at the

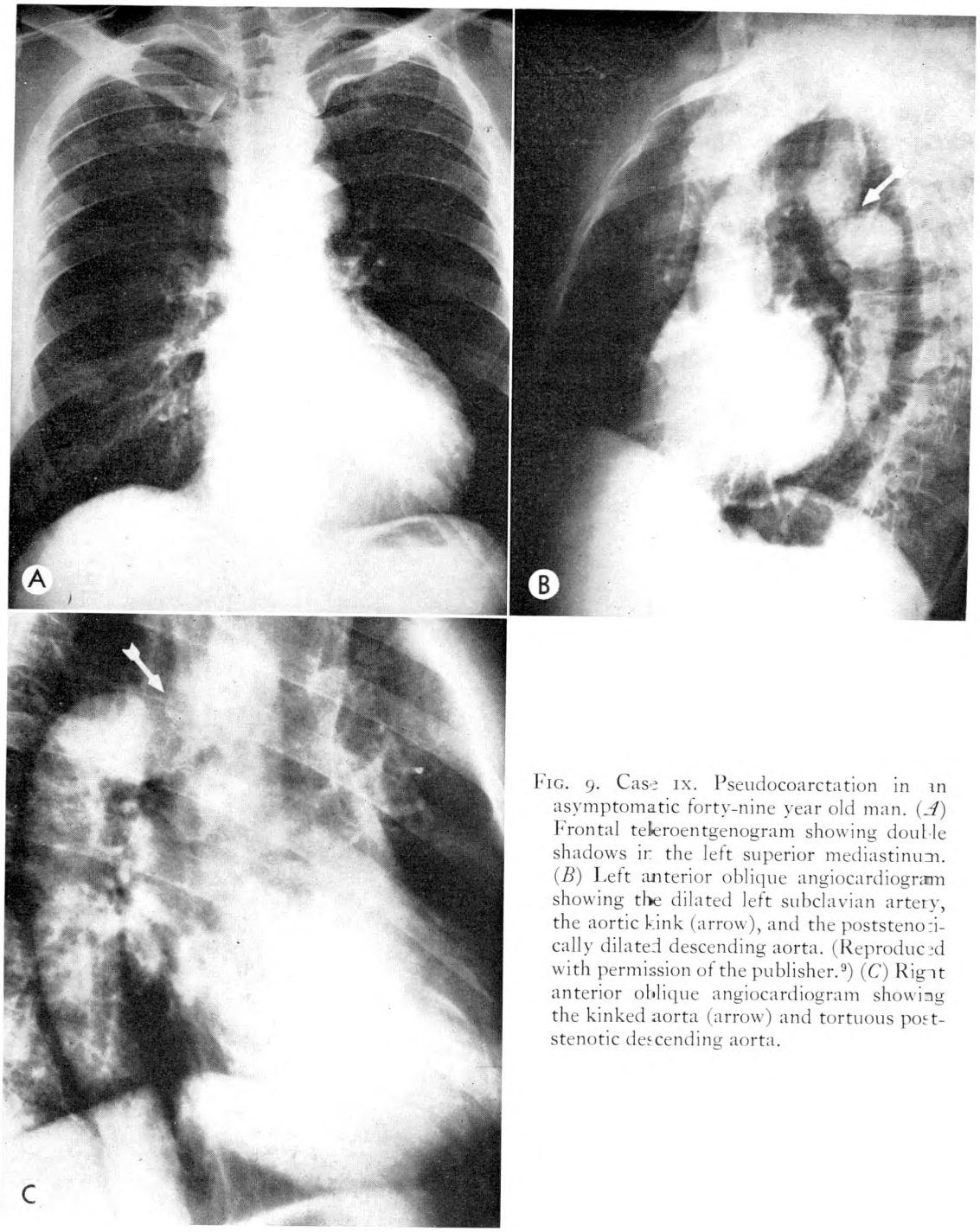


Fig. 9. Case ix. Pseudocoarctation in an asymptomatic forty-nine year old man. (*A*) Frontal teleroentgenogram showing double shadows in the left superior mediastinum. (*B*) Left anterior oblique angiocardiogram showing the dilated left subclavian artery, the aortic kink (arrow), and the poststenotically dilated descending aorta. (Reproduced with permission of the publisher.[9]) (*C*) Right anterior oblique angiocardiogram showing the kinked aorta (arrow) and tortuous poststenotic descending aorta.

point of insertion of the ligamentum arteriosum. This explanation seems more plausible than the one which attributes the aortic arch anomaly to a short, taut, ligamentum arteriosum; elongated aortic arches and left subclavian arteries have been demonstrated in some of the cases in this series.

Dalith[7] has recently emphasized that calcifications below the arch of the aorta

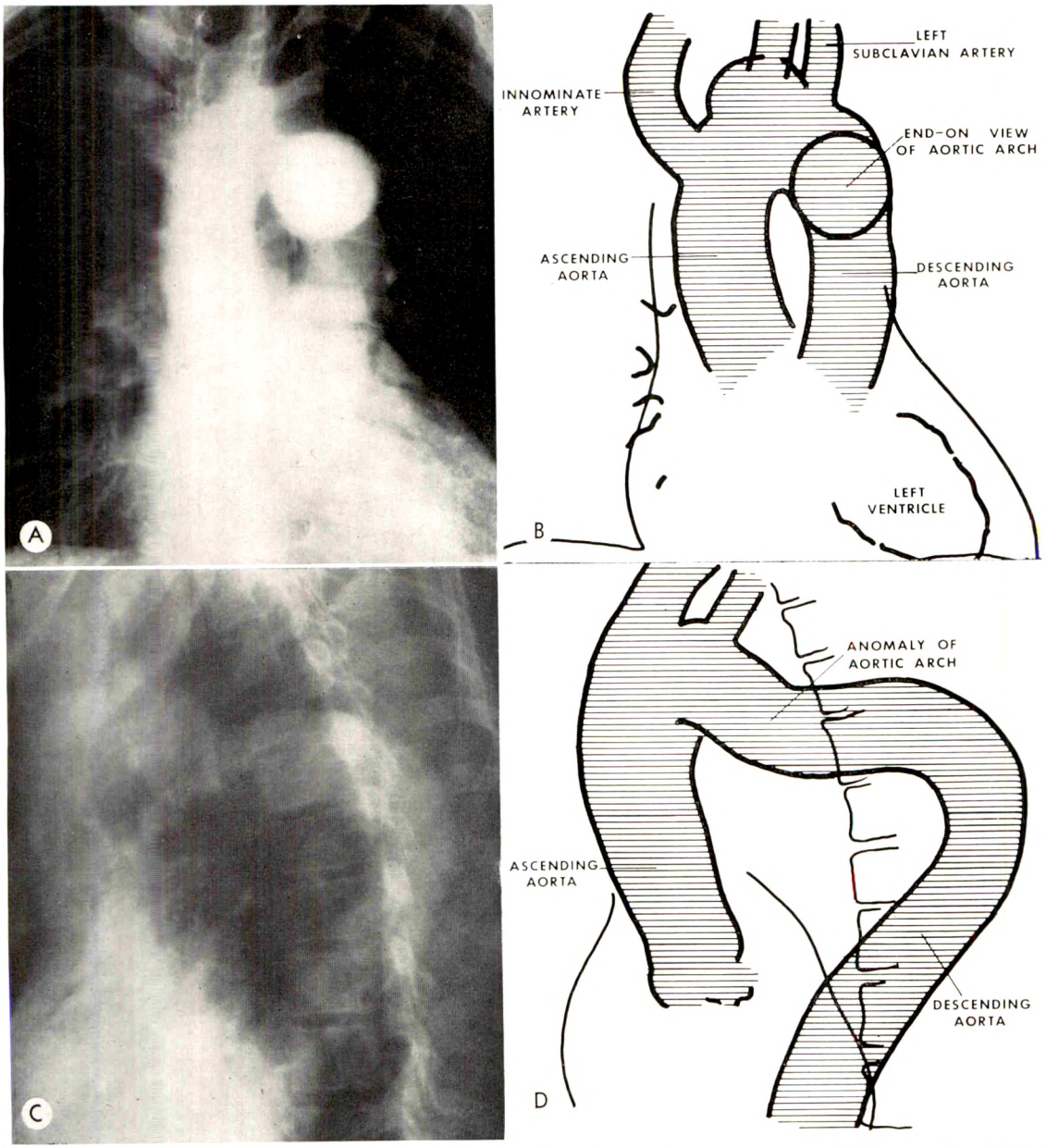


Fig. 10. Case x. Pseudocoarctation of the arch of the aorta in a thirty-seven year old Negress who died of bacterial endocarditis which had perforated the aortic valve seven years after these roentgen studies. (*A*) Frontal angiocardiogram showing an "end on" image of the arch of the aorta. (*B*) Tracing of *A*. (*C*) Left anterior oblique angiocardiogram revealing that the kinked arch of the aorta, with severe tortuosity of the dilated descending portion, was responsible for the peculiar appearance of the arch in *A*. (*D*) Tracing of *C*. (Reproduced with permission of the publisher.[9])

are related to calcific deposits at the sites of insertion of the fifth and sixth embryonic aortic arches. This is verified by the frequent occurrence of calcific deposits in the poststenotic region of the descending aorta in this series (Fig. 2, *A–D*; 3*B*; and 5, *A–D*).

Clinical and Pathologic Findings in the Present Series

Analysis of the clinical data for our 16 patients with deformity of the arch of the aorta (pseudocoarctation) (Table 1) reveals that the sex incidence was nearly

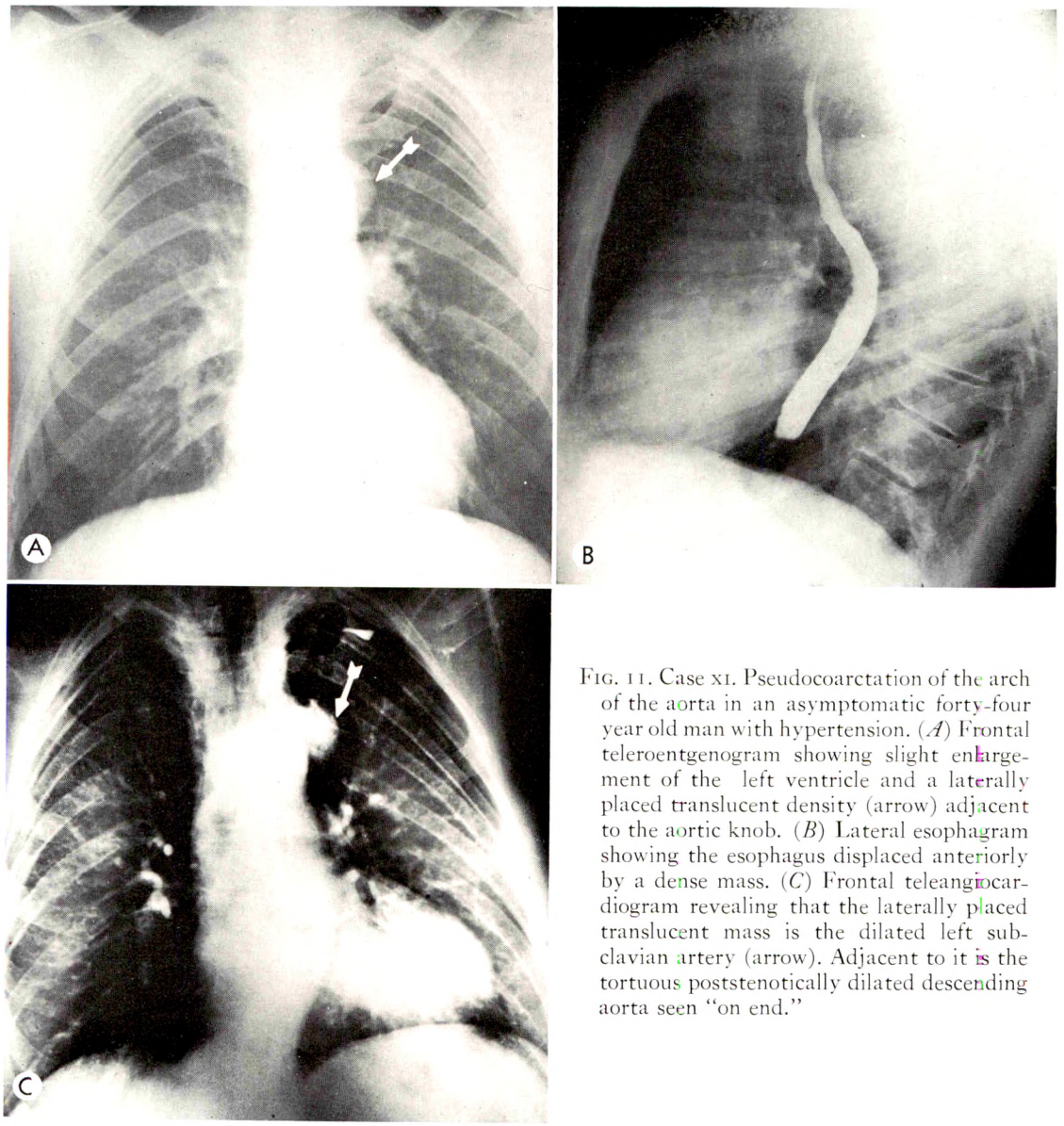


Fig. 11. Case xi. Pseudocoarctation of the arch of the aorta in an asymptomatic forty-four year old man with hypertension. (*A*) Frontal teleroentgenogram showing slight enlargement of the left ventricle and a laterally placed translucent density (arrow) adjacent to the aortic knob. (*B*) Lateral esophagram showing the esophagus displaced anteriorly by a dense mass. (*C*) Frontal teleangiocardiogram revealing that the laterally placed translucent mass is the dilated left subclavian artery (arrow). Adjacent to it is the tortuous poststenotically dilated descending aorta seen "on end."

equal: 9 were males and 7 females. All but 1 were Caucasian (Case x was a Negress). The ages at the time of diagnosis varied from 10 to 58 years (average, 35.5 years). All patients, except the 1 who had heart failure owing to fibroelastosis (Case xvi), were asymptomatic at the time of diagnosis.

Associated with the pseudocoarctation were unrelated conditions such as cervical osteoarthritis and pulmonary emphysema (Case 1), hypertension (Cases ii and xi), and late latent syphilis (Case iii). One patient developed bacterial endocarditis seven years after the angiocardiographic diagnosis of pseudocoarctation, and, at postmortem examination the following year, it was found that the aortic valve had perforated and had caused intractable heart failure (Case x). One patient in the series had aneurysmal dilatation of the aortic sinuses (Case xii). A similar case is recorded in the literature.[12] Four patients had associated congenital aortic valvular stenosis, and

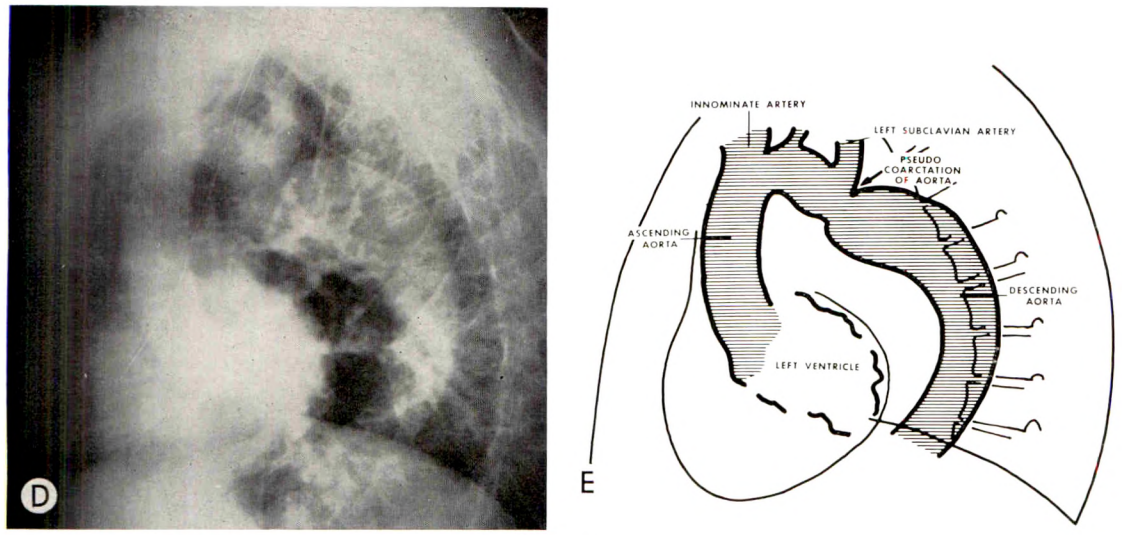


Fig. 11. (*D*) Left anterior oblique teleangiocardiogram showing the markedly dilated left subclavian artery with a kink below it, and a hugely dilated, tortuous, poststenotic descending aorta. (*E*) Tracing of *D*. (*D* and *E* are reproduced with permission of the publisher.[9])

in 2 of these the aortic valve was bicuspid (Cases XIII and XVI). Postmortem examination in the latter case revealed that parietal endocardial fibrosis and fibroelastosis had caused heart failure.[22] Unique in the series is the patient with corrected transposition of the great vessels, dextrorotation of the heart, and eventration of the left diaphragm (Case VIII). The latter malformations apparently occurred at the time of rotational changes that also resulted in pseudocoarctation.

The 4 patients with aortic valvular stenosis and pseudocoarctation of the aorta had the classic systolic murmurs at the base of the heart (Cases XIII–XVI). Seven other patients (Cases II–V, VIII, IX and XII) also had basal systolic murmurs. One patient had in addition an apical systolic murmur (Case II). Bruwer and Burchell[5] and Bruwer[6] have attributed loud basal cardiac systolic murmurs to hemic turbulence through the kinked arch of the aorta. The findings of bicuspid aortic valves in 2 patients (Cases XIII and XVI) and an aortic sinus aneurysm in another (Case XII) suggest that aortic valvular malformations also may be factors in the production of such murmurs. Indeed, Bentivoglio and colleagues[3] and Edwards[11] have written that bicuspid valves are never normal because they tend to become traumatized, fused, stenotic and finally calcified.

Roentgen Findings

Conventional Roentgen Study. This procedure, especially roentgenoscopy, contributes significantly to the diagnosis of pseudocoarctation. In the frontal view, there is an upper, rounded, pulsatile shadow, somewhat translucent because it is surrounded by lung owing to its position high in the left superior mediastinum (Fig. 1, *A* and *B*; 2*A*; 3*A*; 4; 9*A*; 11*A*; and 12, *A* and *B*). Immediately below, there is a more opaque rounded shadow which is usually upwardly convex and which begins just beyond or at the origin of the left subclavian artery. This vessel is dilated and tortuous and resembles the poststenotic dilatation of the descending aorta of a true coarctation. The left oblique and lateral views are often rewarding in revealing that the dense dilated lower shadow is often calcium-lined, thus readily identifying the descending aorta (Fig. 1*C*; 2*B*; 11*B*; and 12*C*). The poststenotically dilated descending aorta also produces indentation of the esophagus which, because of its prominence, especially in the frontal view, may be mistaken for the

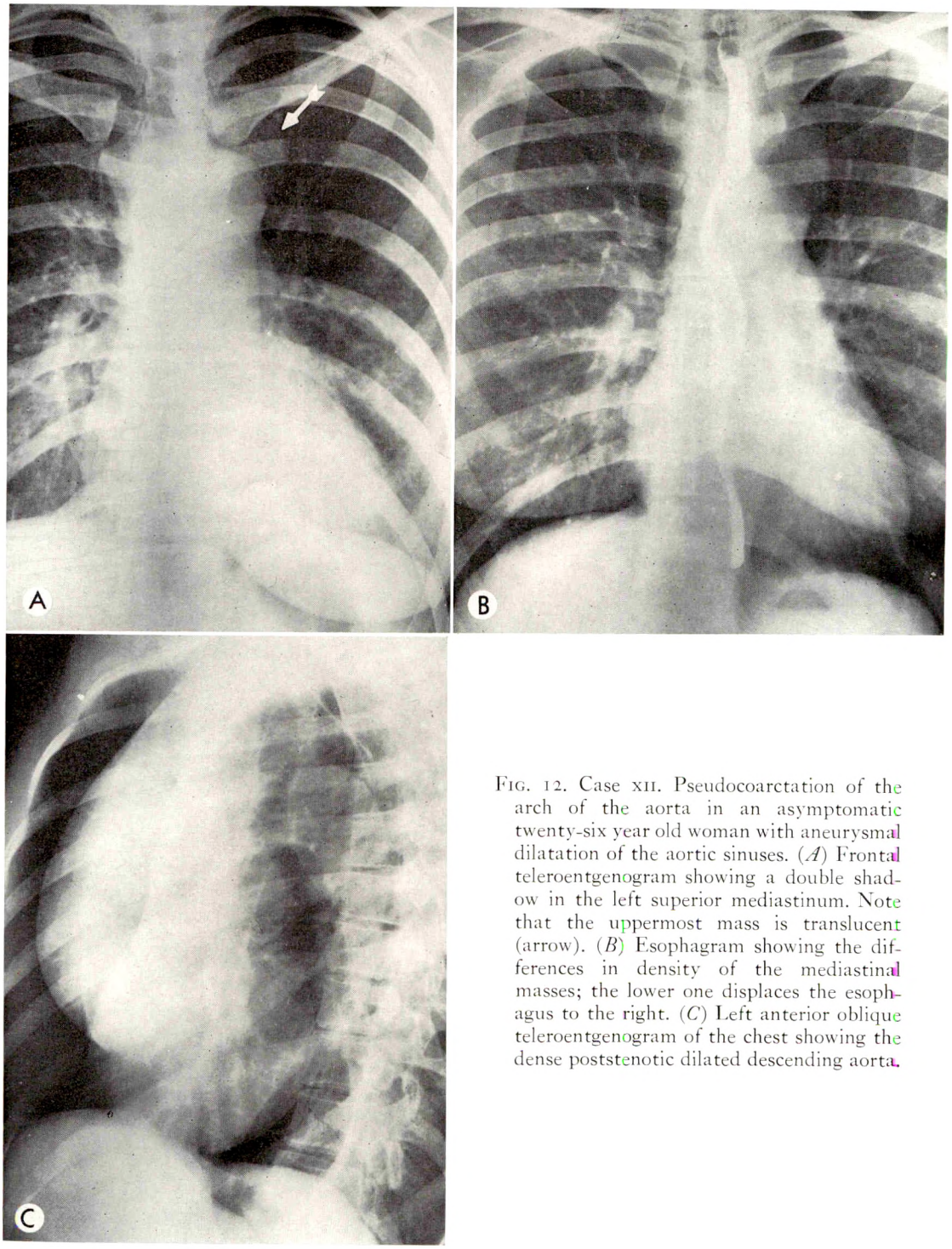


Fig. 12. Case xii. Pseudocoarctation of the arch of the aorta in an asymptomatic twenty-six year old woman with aneurysmal dilatation of the aortic sinuses. (*A*) Frontal teleroentgenogram showing a double shadow in the left superior mediastinum. Note that the uppermost mass is translucent (arrow). (*B*) Esophagram showing the differences in density of the mediastinal masses; the lower one displaces the esophagus to the right. (*C*) Left anterior oblique teleroentgenogram of the chest showing the dense poststenotic dilated descending aorta.

real aortic knob (Fig. 1, *B* and *C*; and 12*B*).

Angiocardiography. This roentgenographic procedure provides the most reliable data regarding anomalies of the arch of the aorta and is far superior to laminagraphy and conventional roentgenography

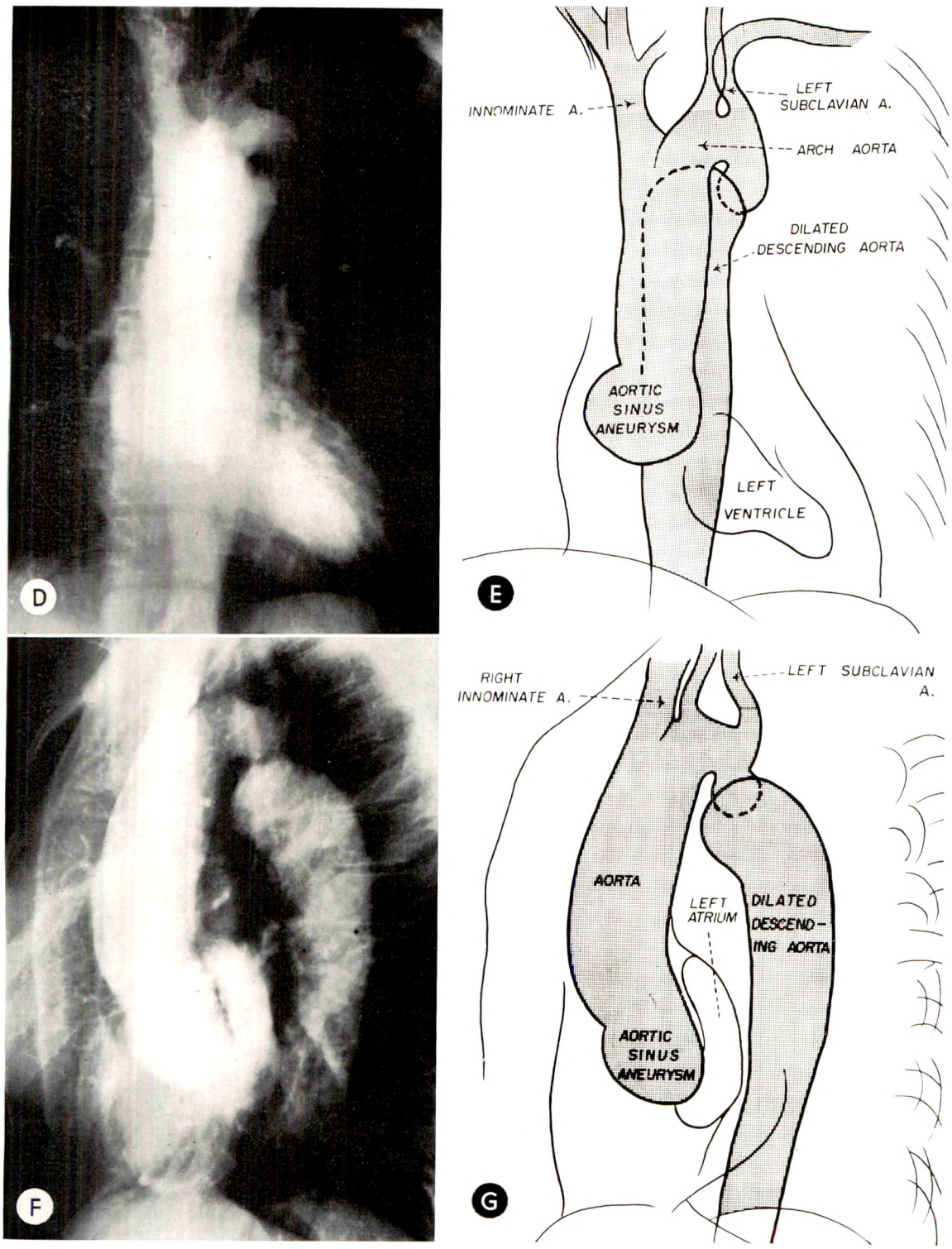


Fig. 12. (*D*) Frontal angiocardiogram revealing the aortic sinus aneurysm, the dilated left subclavian artery, and the poststenotically widened descending aorta. (*E*) Tracing of *D*. (*F*) Left anterior oblique angiocardiogram revealing dilatation of the ascending aorta and left subclavian artery. Note the kink just below the origin of the left subclavian artery and the poststenotically dilated descending aorta. There are no collateral arteries. (*G*) Tracing of *F*. (Reproduced with permission of the *British Heart Journal*.[18])

because opacification of the arch clearly identifies the structures that are concerned in the aortic deformity. Thus, association with lesions such as a sinus of Valsalva aneurysm (Fig. 12, *D–G*) corrected transposition and dextrorotation of the heart (Fig. 8, *B* and *C*) and aortic valvular deformities with poststenotic dilatation of the ascending aorta (Cases XIII–XVI)[22] can be readily demonstrated.

DIFFERENTIAL DIAGNOSIS

The differentiation of pseudocoarctation from true coarctation depends upon the roentgen study. Indeed, the absence of collateral arteries, especially on angiocardiograms, may be the only finding which favors the diagnosis of pseudocoarctation. Hemodynamic data showing absence of hypertension in the arms and legs are then necessary to establish the diagnosis definitively. The conventional frontal roentgenogram, owing to prominence of the malformed aortic arch, may give the impression of a tumor in the left superior mediastinum. The literature contains several references to cases in which exploratory thoracotomy and radiation therapy were carried out because of such a mistaken diagnosis.[13,17]

Aneurysms of the arch of the aorta, especially those due to arteriosclerosis and trauma, also need to be differentiated from pseudocoarctation. The arteriosclerotic aneurysms, usually sacciform or dissecting, are not difficult to diagnose because of their large size and the linear calcific material in their walls. In the case of dissecting aneurysm, a double channel may be identified.[21] The patients with traumatic aneurysms at the top of the aorta usually have a clear-cut history of severe trauma.[19,20]

Other anomalies of the aortic arch are usually associated with constricting rings which cause dyspnea or dysphagia.[4] A diverticulum in a right aortic arch can easily be identified by esophagraphy and, if necessary, by angiocardiographic studies.[14] Tumors in the superior mediastinum may sometimes have transmitted pulsations owing to the proximity of the nearby arch of the aorta and, in such instances, angiocardiography will readily establish the diagnosis.

The diagnosis of pseudocoarctation is not merely of academic interest. The development of bacterial endocarditis in the aortic valve in Case X, seven years after the diagnosis of congenital malformation of the arch of the aorta, proved fatal. The finding of a bicuspid valve in 1 patient at aortic valvulotomy for congenital aortic stenosis (Case XIII) and at postmortem examination in another (Case XVI) underscores the fact that multiple congenital lesions may occur with pseudocoarctation. Further proof is the demonstration of aneurysmal dilatation of the aortic sinuses in Case XII. Case VIII with several congenital cardiovascular anomalies, all apparently occurring at the time of embryonic development of the pseudocoarctation, is still another example of the tendency for multiple congenital cardiovascular anomalies to coexist. Finally, since aortic occlusion is absent in pseudocoarctation (verified by postmortem examination in Case XVI), the presence of a "poststenotic" descending aorta suggests that such a malformation is probably also an associated lesion. Accordingly, it would appear that patients, even with isolated pseudocoarctation, ought to receive antibiotic or chemical prophylactic therapy whenever dental or surgical procedures are contemplated.

SUMMARY AND CONCLUSIONS

Pseudocoarctation of the arch of the aorta is an uncommon congenital anomaly which is anatomically similar to coarctation except that there is no aortic occlusion. Consequently, hemodynamic abnormalities do not occur and the arm and leg blood pressures are normal; collateral circulation does not develop. On the conventional roentgenogram, there is a double rounded shadow along the left superior mediastinum. The upper mass, although translucent because of its high position in the thorax, may be mistaken for a mediastinal tumor or aneurysm; the lower one may be

interpreted as being the aortic knob. Angiocardiography, however, readily delineates the unusually dilated and deformed arch of the aorta and demonstrates that the lower shadow is actually the dilated ("poststenotic") descending aorta.

Some authors have preferred to call this anomaly of the arch of the aorta "kinking" or "buckling". However, such designations do not clearly differentiate this anomaly from true coarctation. Dilatation of the descending portion of the aorta, called poststenotic dilatation in the case of coarctation, also occurs in pseudocoarctation despite the absence of aortic occlusion. Accordingly, it would appear that dilatation of the descending aorta in pseudocoarctation is an additional feature of the malformation.

Heretofore, except for 2 patients with aneurysmal dilatation of the aortic sinuses, associated anomalies of the aorta have not been reported in pseudocoarctation. In 4 patients with congenital aortic stenosis and pseudocoarctation, 2 had bicuspid aortic valves. The first, in a twelve year old boy, was encountered at operation for aortic stenosis; the other case was found at postmortem examination in an adult who died of heart failure owing to parietal endocardial fibrosis and fibroelastosis. Still another patient with pseudocoarctation of the arch of the aorta had corrected transposition of the great vessels and dextrorotation of the heart.

Accordingly, when murmurs at the base of the heart are heard in patients with pseudocoarctation, a bicuspid aortic valve as well as turbulance owing to kinking of the aorta should be suspected. More recent evidence supports the belief that a bicuspid aortic valve is never a normal finding and has a tendency, because of stress, to become roughened, adherent, calcific and stenotic, and is sometimes the site of bacterial endocarditis.

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ADDENDUM

After completion of this paper, another case of pseudocoarctation of the arch of the aorta ("kinking") was found in the recent literature (Šarić, S., Vuletić, V., Gvozdanović, V., and Mark, B. Case of kinking of aortic arch. *Circulation*, 1960, *21*, 1147–1149). Stecken, A., Beyer, A., and Eribo, O. have also recently published a paper on this subject and reported 5 cases (Kinking of the aortic arch [arcus aortae bicurvatus] and "forme fruste" of aortic isthmic stenosis. *Fortschr. Röntgenstr.*, 1961, *94*, 333; abstracted, *Circulation*, 1962, *25*, 903).

Three new patients with pseudocoarctation have also been studied at this center. Two of these have had postmortem examinations and the findings are being prepared for publication (Hagstrom, J. W. C., and Steinberg, I. The pathological lesion in pseudocoarctation of the arch of the aorta).

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VOL. 88, NO. 1

PHYSICAL ASPECTS OF HIGH PRESSURE ANGIOGRAPHY*

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FOR quite some time we have been interested in several factors that might improve the angiographic services in our hospital. This report covers observations that we have made during the development of a high pressure angiographic injection system.

APPARATUS

INJECTORS

The injector[4] with which we have had the most experience is illustrated in the foreground in Figure 1. This injector developes a thrust of 445 p.s.i. (lbs. per sq. in.) hydraulic pressure by using compressed nitrogen. The experimental injector in the background is a modification of the original design so that it develops 1,125 p.s.i. hydraulic pressure. This is accomplished by increasing the mechanical advantage of the system from 1.7 for the original injector to 4.5 for the experimental model.

The mechanical advantage of the system is derived from Pascal's law and is illustrated in Figure 2. P_1 is the applied or gas gauge pressure. A_1 is the effective area of the air cylinder. A_2 is the area of the syringe plunger face. P_2 is the hydraulic pressure. A_1/A_2 defines the mechanical advantage of the system. The hydraulic pressure (P_2) best describes the performance in which we are interested. All charts published should express pressures as hydraulic pressure for the sake of uniformity and clarity to users.

SYRINGE

Our first problem with the new high pressure injector was the development of a safe metal syringe for horizontal use. Figure 3 illustrates a combination hard chrome and stainless steel syringe of satisfactory design. The problem of evacuating air from the nontransparent syringe was

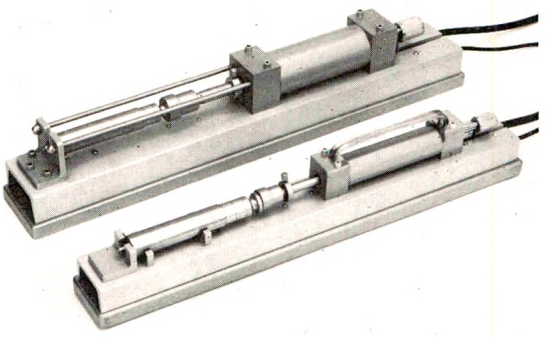
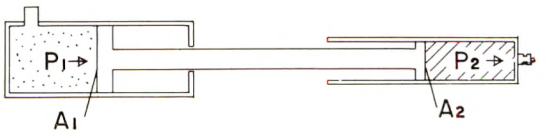


FIG. 1. Injectors with (bottom) 445 p.s.i. and (top) 1,125 p.s.i. hydraulic pressure.

solved by two features in design and one rule of use. The drawing in Figure 3 shows that the end of the syringe barrel has a greater taper than conventional syringes so that air is not trapped about the circumference of the barrel when one holds it vertically to evacuate air. As a second safeguard the plunger tip projects, forming a stop that prevents complete emptying. The rule of loading the syringe concerns the evacuation of air by holding the syringe vertically and injecting into a high pressure transparent connecting tube. The tube is not removed from the syringe. Should the plunger be retracted before the connecting tube is fixed to a catheter, the entry of air is quite apparent in the transparent connecting tube. We do not make a direct



$$P_2 = \frac{A_1}{A_2} \times P_1$$

FIG. 2. Mechanical advantage of the system based on Pascal's law.

* Presented at the Sixty-second Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 26–29, 1961.

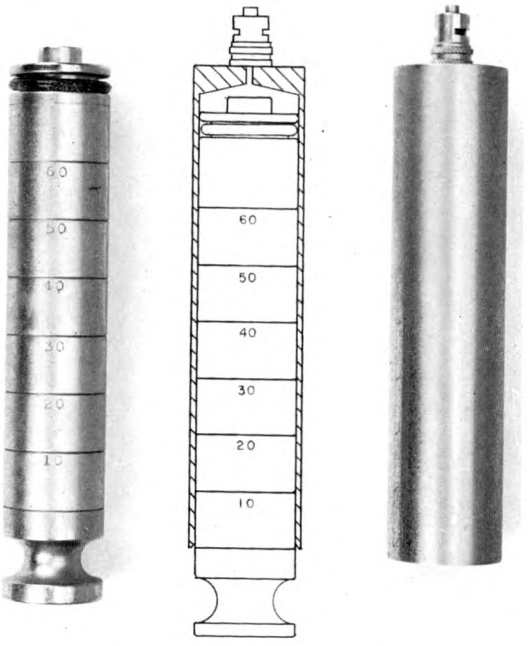


FIG. 3. Stainless steel and chrome syringe.

connection between the connecting tube and the catheter, but interpose a Luer type 3-way stop-cock. This is used to constantly flush the catheter with saline. It required but one serious embolization to convince us of the importance of this point.

STRENGTH OF CATHETER MATERIALS

Quite early it became evident that we needed specific knowledge of the strength of catheter materials in order to avoid untimely breakage.

Figure 4 is a plot of a variety of catheter materials of nearly equal inside diameter (*i.e.*, .066 to .068 inch). These catheters are each 100 cm. in length. These curves were prepared by repeated injections of 50 cc. of water at increasing pressures until the catheters ruptured or until 1,125 p.s.i. hydraulic pressure was reached. The curves are separated only for clarity. The dashed portions represent average ranges of rupture.

Supermid was at first attractive because of its very thin wall. It fails at low pressure by an aneurysmal dilatation or by long rents.

Polyethylene 240 holds up fairly well to 445 p.s.i. hydraulic pressure. Injury to any catheter by sharp bending or clamping with a hemostat begins a shearing effect that will eventually lead to rupture at a low pressure.

The USCI 8F catheter is typical of woven nylon. The wall thickness and strength of the material is greater than PE 240 with the same inside diameter. The performance is better. Some age factor was noted in that older catheters ruptured at a lower position in the illustrated range.

Breakage of a new catheter usually takes place about one-quarter inch from the proximal fitting. Rheologists explain that in turbulent flow there is a given point of maximum stress approximately 50 diameters from the proximal end of a tube and that distal to this a decreasing gradient of pressure exists. This suggested to us that instead of equal wall thickness throughout the length of a catheter, a tapered catheter might be more efficiently constructed. We would therefore have the greatest strength proximally where it is needed.

The U. S. Catheter and Instrument Corporation supplied us with such catheters, made of teflon. By trial we found that the wall thickness of teflon could be reduced to dimensions similar to PE 240 (*i.e.*, outside diameter of 7.3F) and still retain strength superior to nylon. Teflon may be auto-

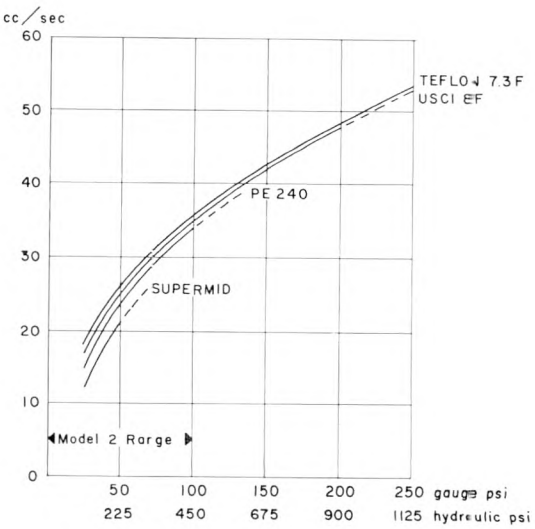


FIG. 4. Catheter material tolerances.

claved. The very low coefficient of friction is quite advantageous in percutaneous insertion.

THE RELATIVE IMPORTANCE OF CATHETER DIMENSIONS, HYDRAULIC PRESSURE AND CHOICE OF CONTRAST AGENT IN THE ANGIOGRAPHIC SYSTEM

Selection of the proper catheter, pressure and viscosity of contrast agent begins with the clinical requirements of the catheter. In our experience the catheter diameters of greatest use are in the French range of 7.3 to 8. However, in Figure 5 we plot the relative viscosity of several contrast agents against the flow rates of a variety of catheters. Each letter on the abscissa, A through L, represents the viscosity of a commonly used contrast agent. To prepare the lower three curves, 50 cc. of the various contrast agents was injected through 100 cm. long No. 7, 8, and 9 French catheters at 445 p.s.i. hydraulic pressure. The top curve, 7.3F, illustrates the performance of the tapered teflon catheter at 1,125 p.s.i. hydraulic pressure. It is apparent that with this 7.3F teflon catheter at 1,125 p.s.i. hydraulic pressure, the flow rate can be increased about 80 per cent over the 8F nylon catheter at 445 p.s.i. Stated another way, if one changes from an 8F catheter (i.d. of .068 inch) at 445 p.s.i. to a 7F catheter (i.d. of .058 inch), then approximately 900 p.s.i. hydraulic pressure is required for equal effluence. However, there is increased probability of rupture because of the inadequacy of the wall thickness of 7F catheters.

In spite of the convenience of the term "flow rate" we are really more interested in opacification. Opacification is the product of flow rate and iodine content and is best expressed in grams of iodine per second injected. Referring to Figure 5 again, the ×'s about the 8F curve represent iodine in grams per second injected through the No. 8 French catheter. These positions in reference to the scale on the right indicate that there is no significant variation in opacification achieved by using the contrast agents of high viscosity. These findings have been

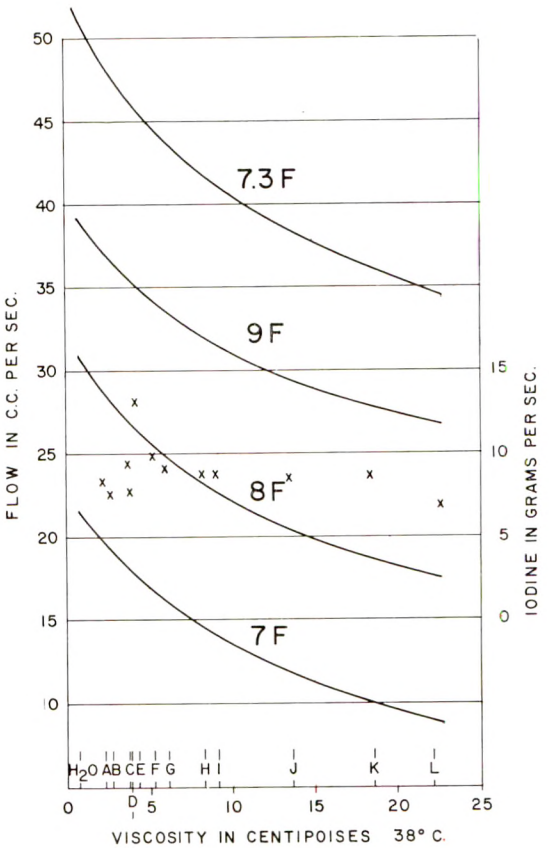


FIG. 5. Relative viscosity of the following contrast agents: A. hypaque 50 per cent; B. miokon 50 per cent; C. diodrast 70 per cent; D. renografin 60 per cent; E. urokon 70 per cent; F. ditriokon; G. renovist; H. hypaque 75 per cent; I. renografin 76 per cent; J. cardiografin 85 per cent; K. hypaque 90 per cent; L. cardiografin 90 per cent.

observed clinically by Dotter and Straube.[2] Lehman and Debbas[3] made similar observations in animal experiments. The use of low viscosity agents therefore obviates the expenditure of time and effort required in heating these concentrated drugs so that they will match the low viscosity agents in opacification.

Because of the large number of factors that may influence the performance of a given angiographic system, we no longer accept performance curves for expected results in clinical use. We have found that the injection time inadvertently may be two to three times that expected. A bent catheter or stopcock partly closed may cause tremendously increased resistance and poor opacification. We now monitor

the performance of each injection by a timer integrated into the injector circuit so that it measures the injection time to one one-hundredth of a second. This timer also permits us to evaluate blood flow, to evaluate planned changes that we make in technique, and it aids us in selecting time corrections in subsequent injections when necessary.

OTHER POINTS FOR CONSIDERATION BY THE ANGIOGRAPHER

It is erroneous to assume that a single feature of a technique, or a particular instrument, will lead to excellence in an angiographic procedure. In evaluating our experiences clinically we have observed four important facets in technical quality that are worthy of emphasis. They are:

1. *The importance of introduction of the contrast agent as near as possible to the site of primary diagnostic interest.* If this is not observed dilution and diffusion of the contrast material may result in poor opacification.

2. *The importance of proper kv. for preservation of contrast.* We have frequently observed that the higher kv. techniques overly penetrate the contrast agent in a vessel as readily as calcium in the bones and, if this fault is not recognized, one may blame the injection system. This problem is evident in large adults. One should work toward the lower kv. techniques for any particular region.

3. *The importance of motion.* Ideally motion should be completely eliminated. For this a sports photographer would recommend exposures of one two-hundredths of a second or less. In practice a 500 ma. generator is our limitation; we need much more improvement, perhaps three to four thousand ma. to obtain very short exposure in adults.

4. *The importance of an adequate injection system.* In five years we have had sufficient experience with the 445 p.s.i. injector to be convinced of its adequacy from the standpoint of both automation and hydraulic pressure. It is capable of completely substituting contrast media for blood in all areas except in the left ventricle and ascending aorta. Here, theoretically, a 1,000 or more p.s.i. injector system may have something to offer. It is our present feeling, however, that the advantages of increasing hydraulic pressure above 445 p.s.i. are limited and are difficult to detect. Future technical improvement in angiography most likely will be made in the area of very short exposures to solve the problems of motion.

SUMMARY

1. The physical features of a new higher pressure injection system have been described. This experimental system develops 1,125 p.s.i. hydraulic pressure. It may prove advantageous in left ventriculography and ascending aortography.

2. Systems developing 1,000 or more p.s.i. hydraulic pressure have limited choice of catheters. A new 7.3F (o.d.) tapered teflon catheter having an i.d. of .068 inch is the strongest and most efficient catheter for use in percutaneous angiography.

3. Data favoring the use of low viscosity contrast agents have been presented.

4. Other technical points of angiography have been emphasized.

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CLINICAL APPLICATIONS OF LYMPHANGIOGRAPHY*

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ROENTGENOGRAPHIC changes in the lymphatic system are readily demonstrated by intralymphatic lymphangiography. Abnormalities of the lymphatics and lymph nodes can be evaluated in the extremities, inguinal, external iliac, common iliac, and para-aortic areas, as well as in the thoracic duct, axillary and supraclavicular regions. This presentation concerns the clinical applications of lymphangiography and includes the results of our experience with 210 patients, 10 of whom were children, the youngest, one year old.

The indications for lymphangiography in our experience are:

1. Evaluation of edema of the extremities.
2. Determination of abnormalities in the thoracic duct manifested by chylothorax and chyloperitoneum.
3. Diagnosis of the gross extent and nature of inflammatory and neoplastic diseases which involve the lymphatic system.
4. As a guide to lymph node dissections.
5. For more accurate portal placement in radiation therapy.
6. As a visual demonstration of the effect of various therapeutic agents.

With the continued accumulation of data, pathologic correlation, and greater technical proficiency in demonstrating other lymph node bearing areas, further applications will undoubtedly be realized.

EDEMA OF THE EXTREMITIES

The patient with edema of an extremity must be evaluated systemically and specific attention must be directed to the affected limb. Arteriography and venography should be done initially to disclose abnormalities in these vessels. Lymphangiography should then be performed bilaterally. The lymphatics should be followed throughout their skeletal pathways to their communication with the subclavian veins, for changes causing edema of the extremities may occur at any intervening point.

Figure 1*A* shows the necessity for demonstrating the lymphatic pathway totally. This was the case of a fifteen year old female patient with bilateral lower extremity edema since infancy. The veins of both legs appeared normal on venograms. Lymphangiograms demonstrated an increase in the number of lymphatics in both extremities. In the retroperitoneal area, there were anomalous lymphatics at L3 and L4 on the right and T11 and T12 on the left, simulating the appearance of cavernous lymphangiomata. In such cases it is possible that this abnormality may be responsible for the production of edema, and if so, surgical treatment might be curative.

Kinmonth *et al.*,[2] in studying idiopathic lymphedema, concluded that congenitally anomalous lymphatics were present in most cases, being manifest following trauma, surgery or other forms of insults. They classified these changes into aplasia, hypoplasia, varicose or dilated lymphatics with dermal backflow, and dermal backflow alone. Our experience is primarily with secondary lymphedema. Thus far, we have seen no instance of aplasia. Hypoplasia of the lymphatics has been most frequently associated with chronic venous disease. Dilated lymphatics have been associated with acute inflammatory episodes in the adjacent structures; for example, acute thrombophlebitis. Dilated lymphatics with dermal backflow or dermal backflow alone have been encountered most frequently with

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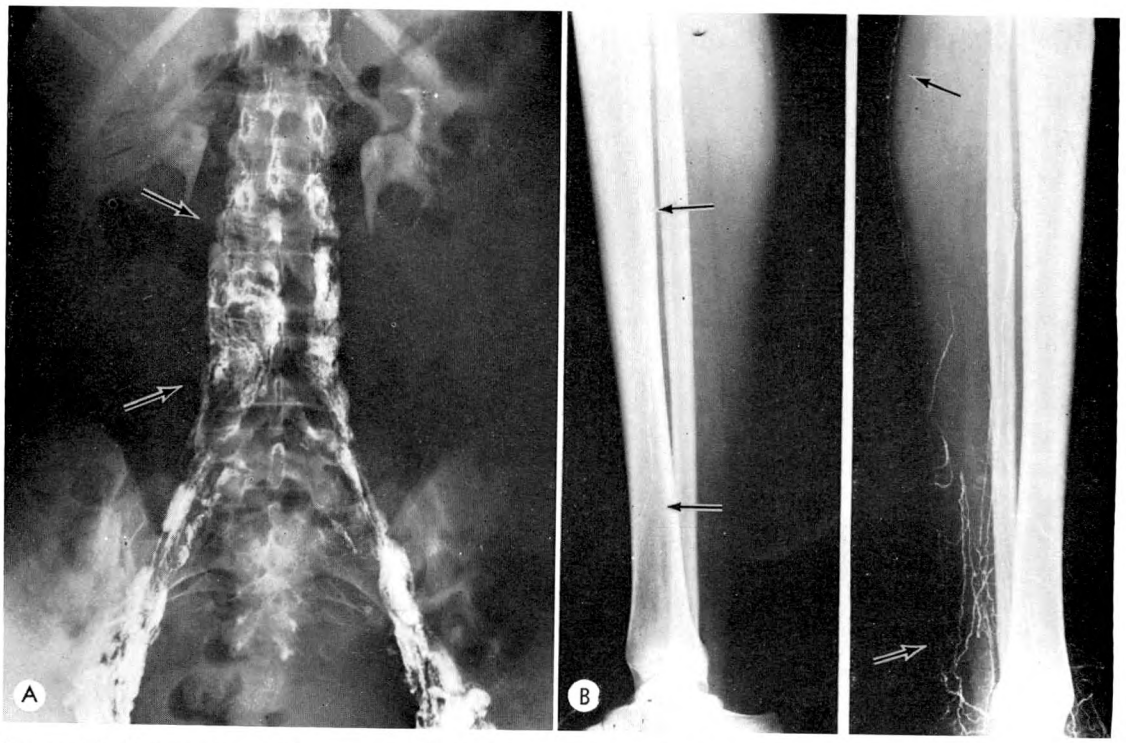


FIG. 1. (*A*) Retroperitoneal lymphatics of a fifteen year old girl with idiopathic lymphedema of both lower extremities. There is a dilated collection of anomalous lymphatics at L_3 and L_4 on the right. (Reproduced with permission from *Radiology*.) (*B*) Idiopathic lymphedema of the lower extremity. The extremity on the left shows a nornal lymphatic pathway while that on the right shows absence of filling of the greater saphenous system with the collateral lymphatics leading to and draining via the lesser saphenous system.

peripheral lymphatic obstruction, either on a congenital or acquired basis (inflammation, surgery, trauma, neoplasm, or post irradiation). Dermal backflow is merely the manifestation of collateral circulation through skin lymphatics secondary to peripheral obstruction. The radiopaque material, which usually empties from the peripheral channels in a few hours, remains for days, demonstrating the marked delay in circulation. We feel that in many cases this is the result of edema, rather than the etiology. The etiology or site of the obstruction may frequently be obscure.

Figure 1*B* shows a patient with edema of the lower extremity of unknown etiology. The normal lymphatic pathway through the greater saphenous distribution was obstructed, and the flow was maintained through the dermal vessels to the lesser saphenous pathway and eventually to the femoral lymph nodes. There was a delay of days in the emptying of these channels.

A common agent in the production of edema is found in relation to inflammatory changes in the veins in both acute and chronic thrombophlebitis. The veins are readily opacified, substantiating the inflammatory changes; however, associated changes may also be seen in the lymphatics. In the acute episode, the lymphatics are dilated and the adjacent lymph nodes are enlarged. This may be due to an increase in the volume of local interstitial fluids or to a partial obstruction secondary to the inflammatory changes in the lymph nodes. With resolution, the lymphatics and lymph nodes revert to normal. In contrast, in chronic thrombophlebitis, the superficial lymphatics are decreased in number and caliber. The lymph nodes remain enlarged. Occasionally, the veins in lymphangiograms are outlined by a sleeve of contrast material, probably in the perivascular lymphatics.

The complication of postmastectomy edema has produced controversy as to whether it is primarily venous or lymphatic in origin. We have found the pattern to be variable, depending on the predominant involvement, ranging from almost pure lymphatic obstruction to primarily venous occlusion (Fig. 2, *A* and *B*). In chronic venous disease, there is a decrease in the number and caliber of lymphatics. On the other hand, in cases of lymphatic obstruction dilated, tortuous lymphatics with dermal backflow are visualized (Fig. 2, *C* and *D*). Between the two extremes are combinations of each showing changes on the lymphangiograms. With greater understanding of these changes, the determination of the clinical approach will prove less difficult.

THORACIC DUCT ABNORMALITIES

The changes in the thoracic duct in certain diseases can now be demonstrated by lymphangiography. In cases of chylothorax, the exact site of the laceration or obstruction in the thoracic duct can be visualized. In a patient with intermittent chylothorax producing marked dyspnea, lymphangiograms revealed the site of obstruction in the thoracic duct (Fig. 3, *A* and *B*). At exploration, a plaque of fibrotic tissue overlay the duct in this area. The plaque was removed, the duct tied, and a talc poudrage performed, relieving the patient's symptoms.

The demonstration of the intercostal lymphatics and mediastinal lymph nodes is brought about by partial obstruction of the thoracic duct by a mediastinal mass or anomalous thoracic pathways. Figure 3*C* shows the intercostal lymphatics in a patient with a superior mediastinal mass previously diagnosed as Hodgkin's sarcoma by supraclavicular lymph node biopsy. The thoracic duct was well outlined along with the collateral channels by way of the intercostal lymphatics. Figure 3*D* shows the normal appearance of the thoracic duct.

EXTENT AND NATURE OF DISEASE

The demonstration of lymph nodes is of extreme value in assessing many diseases. Normal lymph nodes have a variable size, averaging approximately 1.5 to 2 cm. in maximum dimension. Each has a distinct margin with possibly one indentation at the hilus. The internal architecture has a homogeneous reticular pattern (Fig. 4*A*). The lymph nodes are best demonstrated on lymphangiograms made twenty-four hours after injection and can be visualized from one month to as long as ten months without further injection. Inflammatory lymph nodes are larger in size but otherwise have a normal appearance. There is frequently an associated increase in the number of lymph nodes but the peripheral contour and architecture are normal.

In lymph nodes involved with metastatic carcinoma, the margins are irregular with a moth-eaten appearance (Fig. 4*B*). When the lymph node is partially replaced by tumor, only the residual functioning portions are demonstrated. With complete replacement by tumor, the lymph nodes will not be visualized and the lymphatics may be seen circumventing these areas. However, with associated complete lymphatic obstruction, secondary evidence of total replacement is present because of the development of collateral lymphatic circulation. Interior vena cavagraphy is done in the event of suggestive total replacement of the nodes by neoplasm.

The pattern of the lymph nodes in cases of lymphoma is characteristic. There is a general increase in the size and in the number of the lymph nodes; however, the borders remain intact. The internal architecture has a foamy or lacy appearance (Fig. 4*C*). At present, it seems that the different microscopic findings separating lymphomas into various groups may be reflected in the patterns observed grossly on the lymphangiograms. The lymphosarcomatous involvement of the lymph nodes is typical. Hodgkin's granuloma shows a similar background pattern but superimposed on this are punched-out defects within the lymph node. Chronic lymphocytic leukemia may vary slightly from the basic

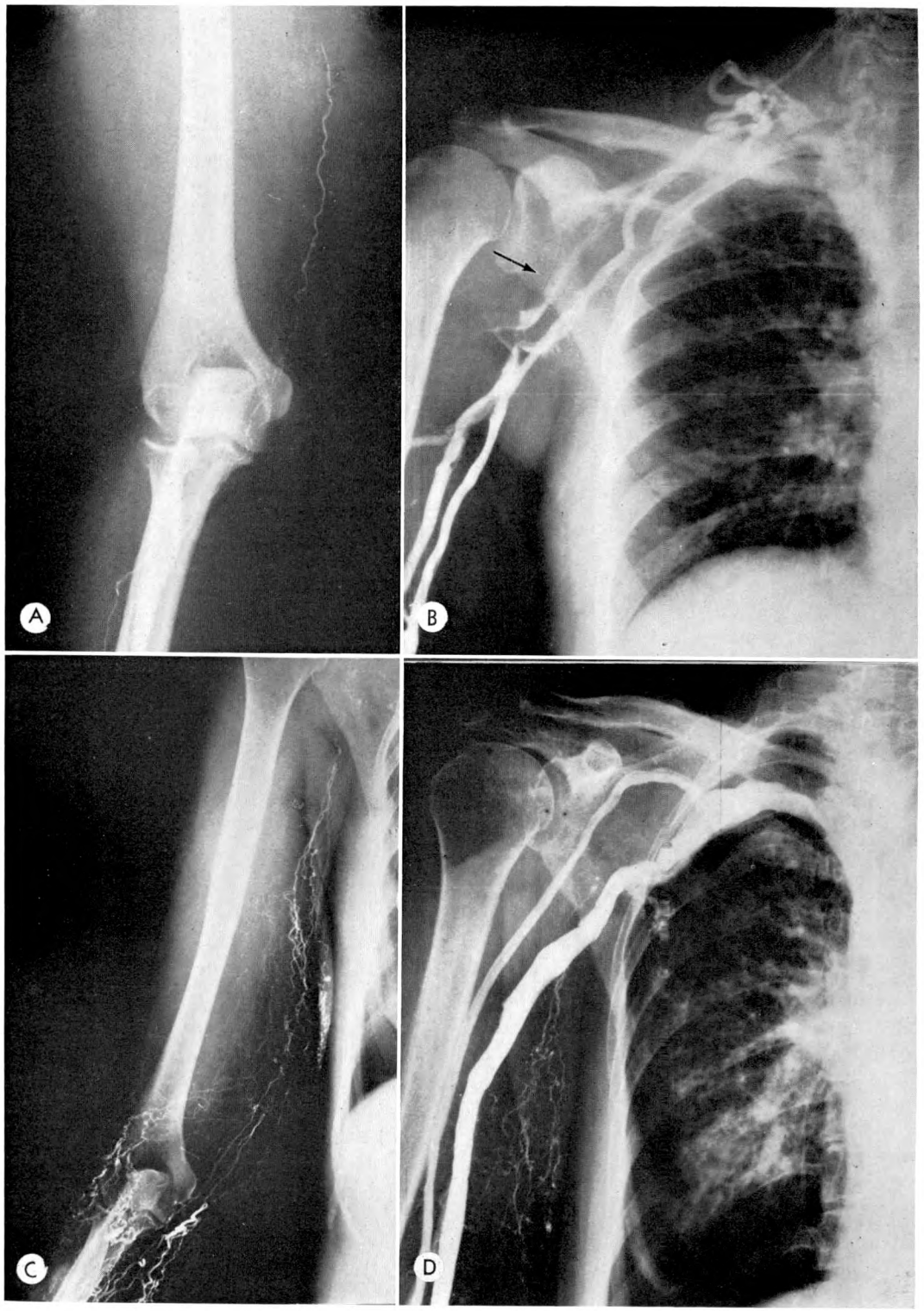


FIG. 2. (*A*) Postmastectomy edema with reduction in the number and caliber of the demonstrated lymphatics (*B*) Venogram of the case shown in *A* demonstrates partial venous obstruction probably due to chronic venous disease. (*C*) Postmastectomy edema with dilated, tortuous lymphatics and dermal backflow. (*D*) Venogram of patient in *C* shows absence of venous disease.

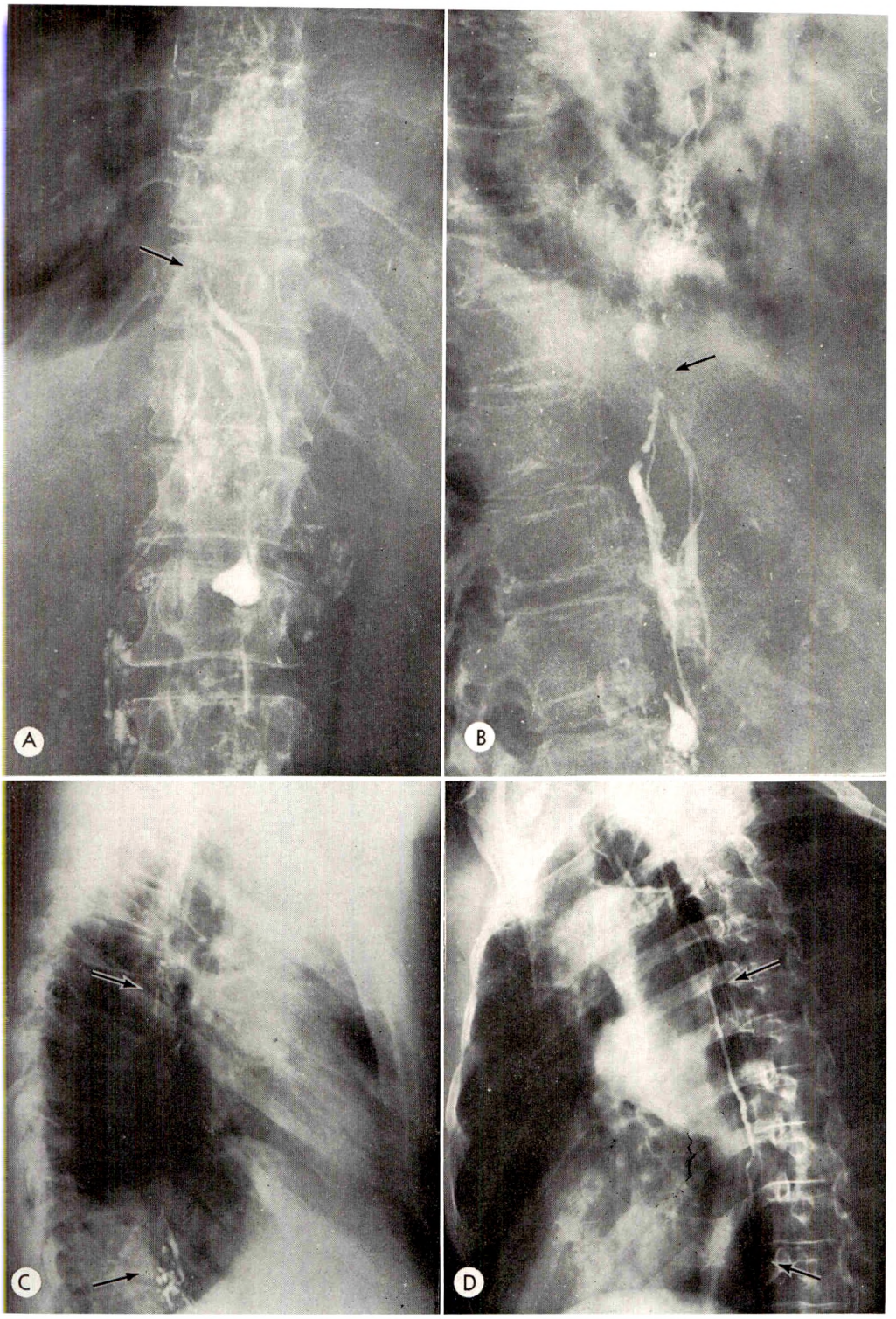


Fig. 3. (*A* and *B*) Patient with bilateral chylothorax presenting obstruction of the thoracic duct. Surgery was performed to remove a fibrotic plaque obstructing the duct at this level, and following this bilateral talc poudrage was done. The patient has had no recurrence of pleural fluid. (*C*) Intercostal lymphatics and collateral lymphatics visualized because of partial obstruction of the thoracic duct due to mediastinal Hodgkin's disease. (*D*) Normal thoracic duct in oblique projection.

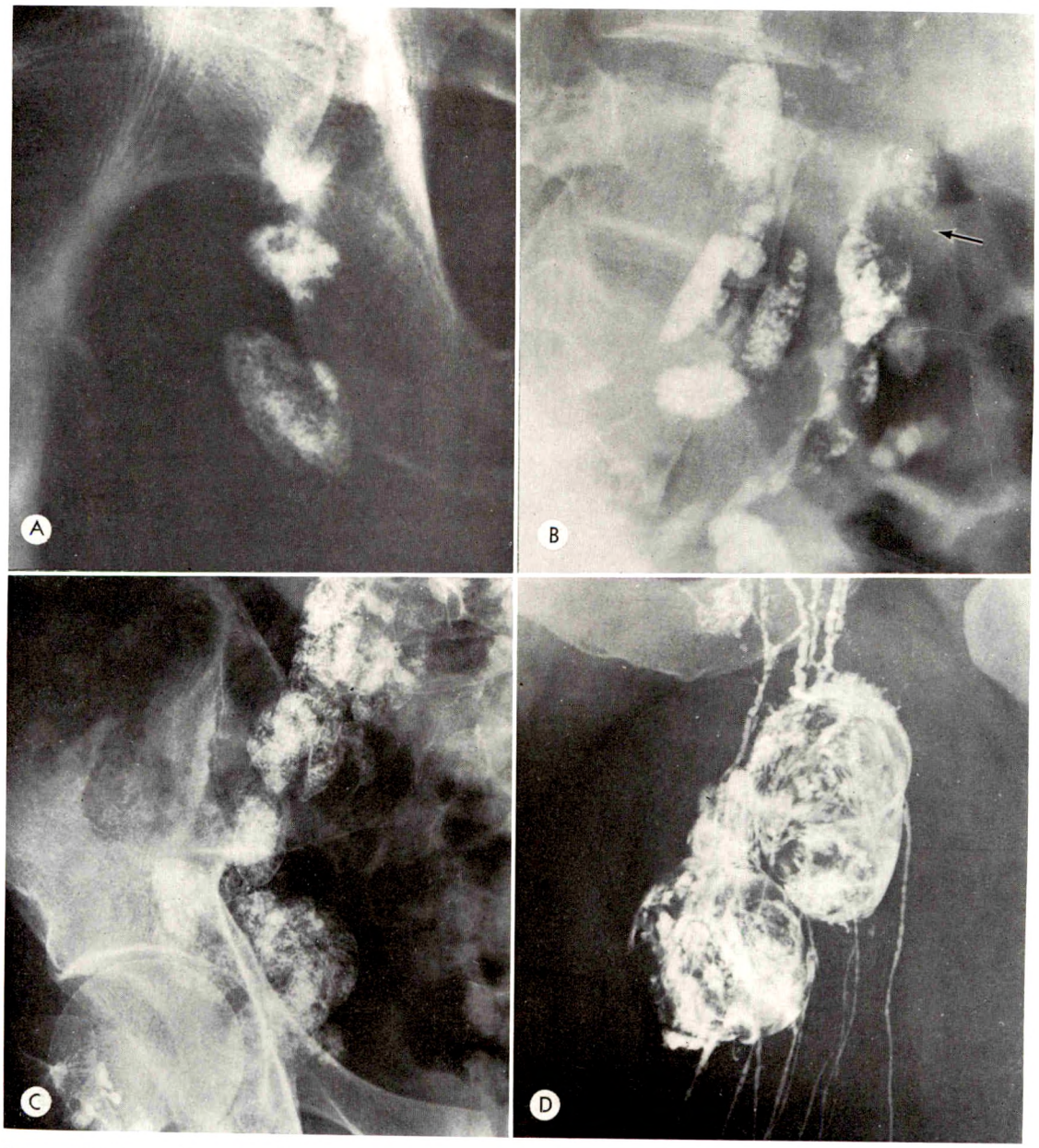


FIG. 4. (*A*) Normal lymph node with regular border and a homogeneous reticular pattern within this border. (*B*) Metastatic carcinoma involving the retroperitoneal lymph nodes in a patient with adenocarcinoma of the rectum. The margin of the lymph node is irregular and moth-eaten. (Reproduced with permission from *Radiology*.) (*C*) Lymphosarcoma presenting enlarged lymph nodes with a regular border but a foamy or lacy internal architecture. (Reproduced with permission from *Radiology*.) (*D*) Malignant melanoma with areas of spherical replacement of the lymph node tissue.

lymphomatous pattern. In the few patients studied to date, there appeared to be flocculent areas of increased density within the lymph nodes. In advanced cases, it is difficult, if not impossible, to differentiate the specific types of lymphomata.

In 4 of our patients with metastatic malignant melanoma involving the lymph nodes, we have observed similar changes (Fig. 4*D*). The metastatic areas presented

a more definite spherical appearance. With further study, this may prove to be a characteristic finding.

False positive findings of lymph node involvement have been encountered. These were due to inadequate filling due to technical difficulties, fibrotic changes (postinfectious, postsurgical, or postradiation therapy), fatty replacement, abscess formation or occasional superimposition of lymph nodes producing a "defect" between them. The lymph node area causing the most confusion has been the inguinal region. These lymph nodes are frequent sites of chronic infection and should not be used as a basis for the evaluation of malignant involvement unless this is the local drainage area for a primary lesion. For generalized diseases, other areas are more reliable.

For completely studying and treating patients with neoplasms, lymphangiography is extremely helpful. This procedure has been useful in determining the gross nature and extent of disease, whether neoplastic or inflammatory. It is even more important in the study of areas not readily palpable, especially the retroperitoneal space, the mediastinum and even the apex of the axilla.

Figure 5*A* shows a patient with carcinoma of the breast who had two palpable axillary lymph nodes, with clinically diagnosed metastatic disease. Lymphangiograms demonstrated many lymph nodes with moth-eaten margins, all of which were suspected to contain metastatic carcinoma. A radical mastectomy was performed with complete removal of all the demonstrated lymph nodes. These lymph nodes were only partially replaced by tumor, while others totally replaced by tumor contained no contrast material and were not visualized.

The direct lymphatic drainage in the female pelvis is to the internal iliac lymph nodes and then to the common iliac chain. Thus far, by lymphangiography we have not demonstrated the immediate visceral drainage through the internal iliac lymph nodes. However, the evaluation of the common iliac pathway is of importance. If complete obstruction occurs, collateral circulation is visualized. For example, a patient with carcinoma of the endometrium was examined by lymphangiography (Fig. 5*B*). Both common iliac lymphatic chains were obstructed and collateral channels were demonstrated. This was considered secondary evidence of metastatic disease in the lymph nodes of the common iliac chains. The changes in the visualized lymph nodes represented metastatic carcinoma.

The extent of disease in the retroperitoneal area, if great enough, can be evaluated by intravenous urography which reveals displacement or distortion of the normal structures. The cause of this distortion and obstruction of the urinary tract may be more specifically determined by lymphangiography. Figure 5*C* shows a patient who was treated for a carcinoma of the rectum by an abdominoperineal resection. A short time later, the patient complained of left flank pain. The intravenous urogram disclosed hydronephrosis and obstruction of the left ureter; the lymphangiogram clearly demonstrated the metastatic carcinoma in a large lymph node producing the obstruction. In another instance distortion of the urinary tract by lymph nodes involved with neoplasm was seen in a patient with lymphosarcoma. The intravenous urogram revealed the displacement of the kidneys; the lymphangiogram demonstrated this abnormality to be due to a mass of lymphosarcomatous lymph nodes (Fig. 5*D*). A similar situation was seen in an eight year old patient with a known diagnosis of lymphosarcoma. The intravenous urogram revealed the obstruction of the right ureter; the lymphangiogram demonstrated this obstruction to be secondary to lymphosarcomatous changes in the retroperitoneal lymph nodes (Fig. 6, *A* and *B*).

"Localized" lymphoma can be evaluated more carefully by lymphangiography. In some patients, complaints not referable to the obvious lesion, such as vague abdominal discomfort, may be found to be secondary to a more generalized process. We have found that lymphomata are seldom local-

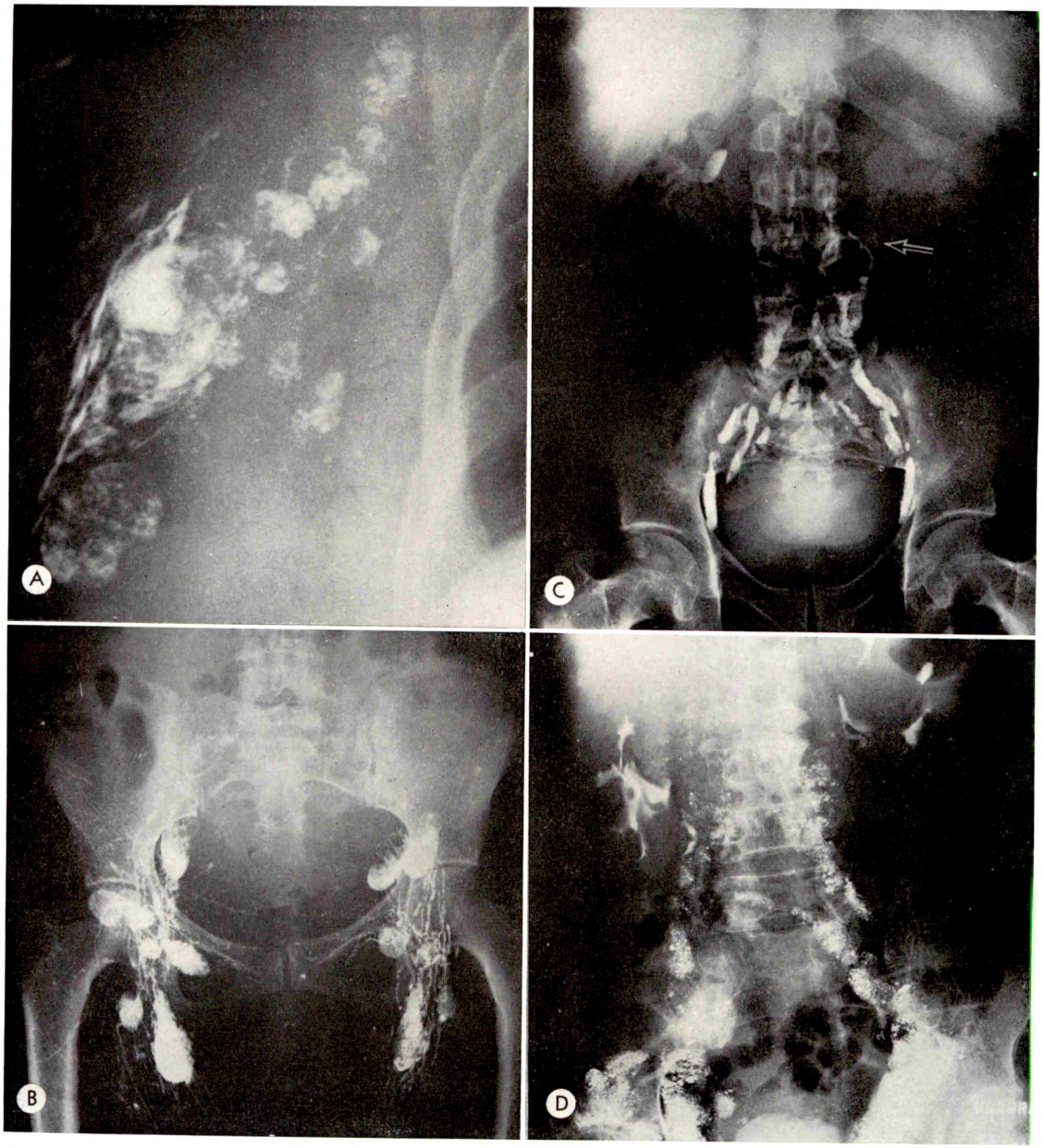


FIG. 5. (*A*) Lymphangiogram of a patient with carcinoma of the breast. Only two lymph nodes were palpable clinically. All lymph nodes were found to be involved with tumor on histologic examination. (Reproduced with permission from *Radiology*.) (*B*) Involvement of the common iliac chains and demonstration of collateral lymphatics in the pelvis of a patient with endometrial carcinoma. This was considered secondary evidence of internal iliac disease. (*C*) Metastatic carcinoma to a lymph node producing ureteral obstruction. (*D*) Displacement of the kidney due to lymphosarcomatous enlargement of the retroperitoneal lymph nodes.

ized. Evaluation of the retroperitoneal area by lymphangiography is most informative. In a patient who had a history of vague abdominal discomfort for one year, all the usual roentgenographic studies failed to disclose definite abnormalities. A supraclavicular lymph node biopsy was diagnostic of Hodgkin's granuloma which

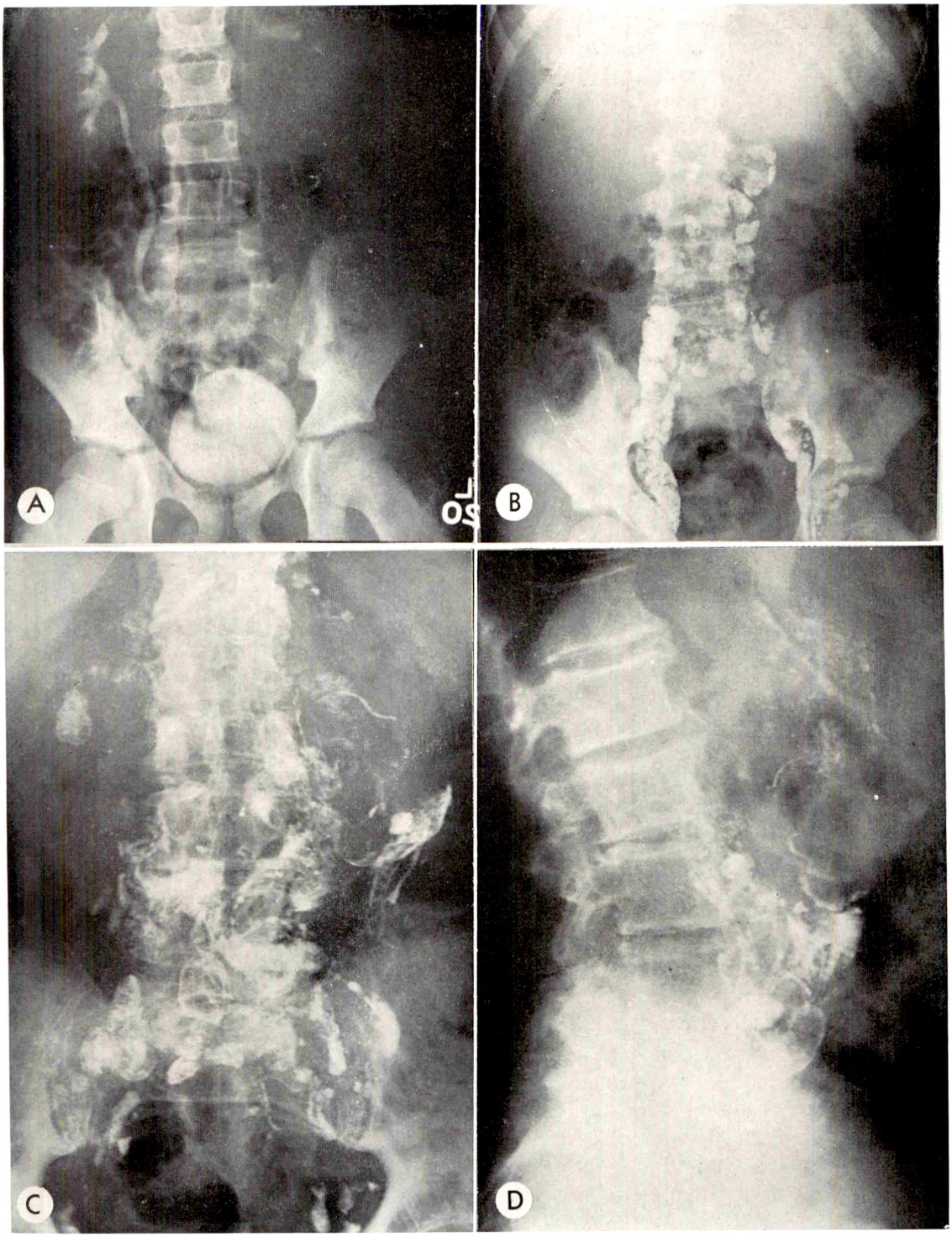


Fig. 6. (*A*) Intravenous urogram and (*B*) lymphangiogram demonstrating right ureteral obstruction due to lymphosarcoma in an eight year old child. Anteroposterior (*C*) and lateral (*D*) roentgenograms of a patient with known cervical Hodgkin's disease and a history of abdominal pain. The lymphangiograms in this case showed huge retroperitoneal lymph nodes which were not palpable. Treatment to this area relieved the patient's symptoms.

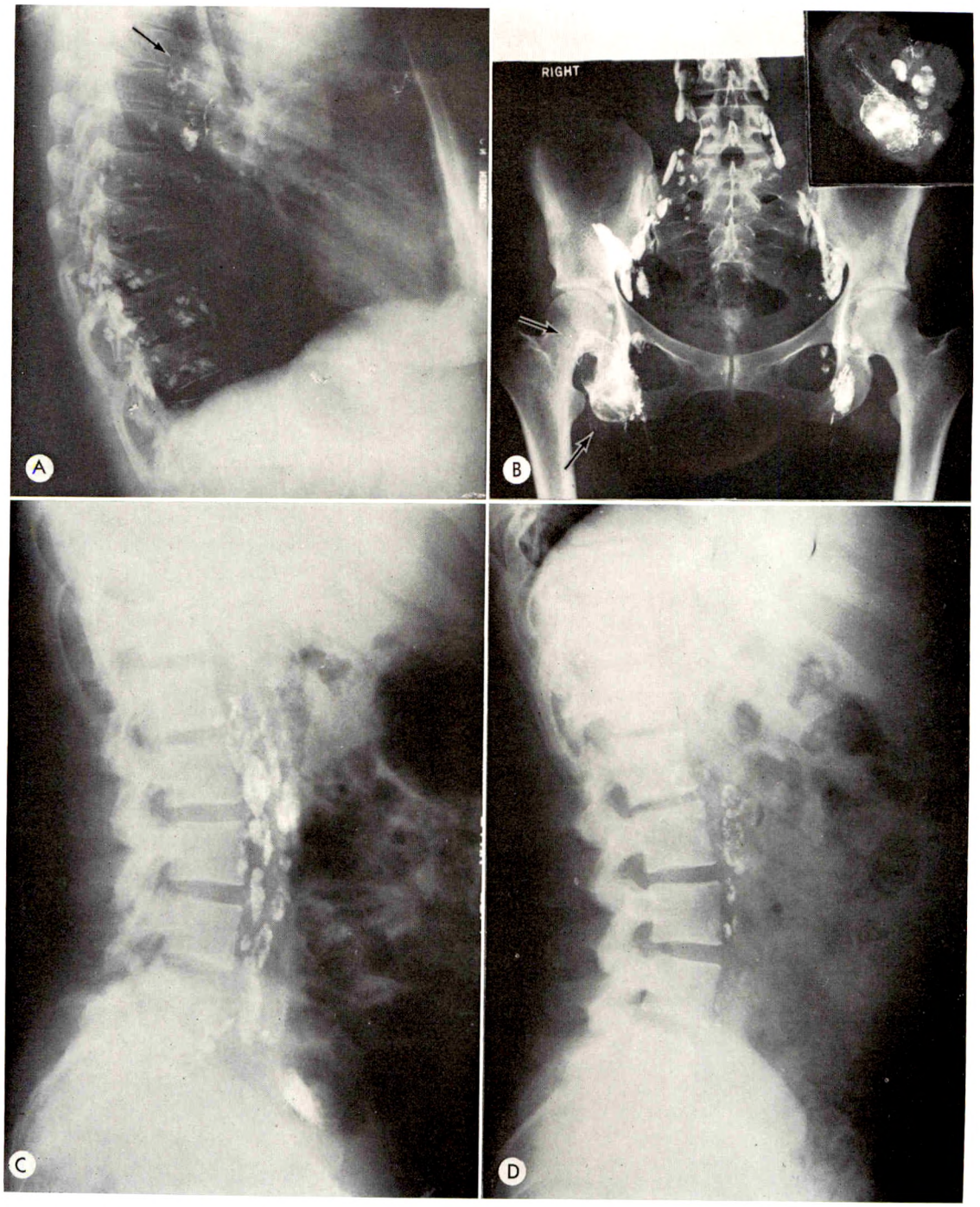


FIG. 7. (*A*) Involvement of the mediastinal lymph nodes with metastatic carcinoma is demonstrated in a patient with a mediastinal mass. These lymph nodes will only fill if the mass partially obstructs the thoracic duct. (*B*) Femoral lymph node mass is seen in a patient with malignant melanoma with no demonstrable metastasis above this point. Surgical removal of the obviously involved mass was performed. Preoperative (*C*) (Reproduced with permission from *Radiology*) and postoperative (*D*) roentgenograms of a patient with metastatic testicular carcinoma. Several of the lymph nodes were left behind after a retroperitoneal dissection.

was thought to be localized. The patient was given local radiation therapy to these lymph nodes. His abdominal complaints continued. Lymphangiograms demonstrated extensive Hodgkin's granuloma in the retroperitoneal area, and treatment by radiation relieved his symptoms (Fig. 6, *C* and *D*).

Other diagnostic problems have been studied by lymphangiography and considerable information has been obtained, especially in evaluating patients with fever of unknown origin and undiagnosed back pain where there is a strong suggestion of malignancy. Occasionally, this study is a valuable diagnostic aid in a patient with a mediastinal mass. If the lesion obstructs the thoracic duct, the mediastinal lymph nodes may be filled with the opaque medium. In one patient the differential diagnosis of a superior mediastinal mass was assisted by lymphangiography which revealed lymph node involvement by metastatic carcinoma (Fig. 7*A*). The final diagnosis was anaplastic carcinoma of the lung.

LYMPHANGIOGRAPHY AS A GUIDE TO LYMPH NODE DISSECTION

Lymphangiography has been utilized by the surgeon to determine the advisability of a lymph node dissection and as a guide to the complete removal of these lymph nodes. Lymphangiograms taken at the time of operation have been of great assistance. This has been of benefit in retroperitoneal lymph node dissection for treatment of teratocarcinoma of the testicle, radical mastectomy with axillary lymph node dissection, and inguinal lymph node dissection for metastatic malignant melanoma, vulvar carcinoma and penile carcinoma (Fig. 7, *B*, *C* and *D*).

LYMPHANGIOGRAPHY IN RADIATION THERAPY

In radiation therapy we have employed lymphangiography for more exact portal placement. The response of lymph nodes to radiation therapy can be more specifically determined, especially in inaccessible areas. Because of the retention of contrast material in the lymph nodes, the course of the disease during and after radiation therapy can be followed.

A diagnosis of Hodgkin's granuloma in a patient was made by lymph node biopsy. He had considerable inguinal, pelvic, and retroperitoneal lymph node involvement. Lymphangiograms taken at the beginning of therapy and then two months later demonstrated the good results of therapy (Fig. 8, *A* and *B*).

LYMPHANGIOGRAPHY IN CHEMOTHERAPY

The therapeutic approach to lymphomatous diseases has been altered somewhat by the study of the extent of lymph node involvement. With diffuse disease, treatment by chemotherapy is often the preferred approach. The effectiveness of such therapy can be more readily assessed with the visualization of the lymph nodes. In a patient with known Hodgkin's granuloma and diffuse peripheral lymphadenopathy, lymphangiography demonstrated the involvement of the retroperitoneal lymph nodes. Following nitrogen mustard therapy, the patient improved clinically. This was reflected on lymphangiograms by the marked decrease in the size of the lymph nodes (Fig. 8, *C* and *D*).

INTRALYMPHATIC INFUSION

Still another avenue utilizing lymphangiography has been the attempt at treatment of neoplastic disease by the intralymphatic infusion of chemotherapeutic agents and radioisotopes. In carcinomatous involvement of the lymph node, the contrast agent may bypass the specific tumor area because only residual functioning tissue is demonstrated by lymphangiography. However, in lymphoma, this approach may be of some value. We have treated 10 patients with little demonstrable success. The chemotherapeutic agent injected should be soluble in oil, thereby assuring its distribution throughout the lymphatic chain.

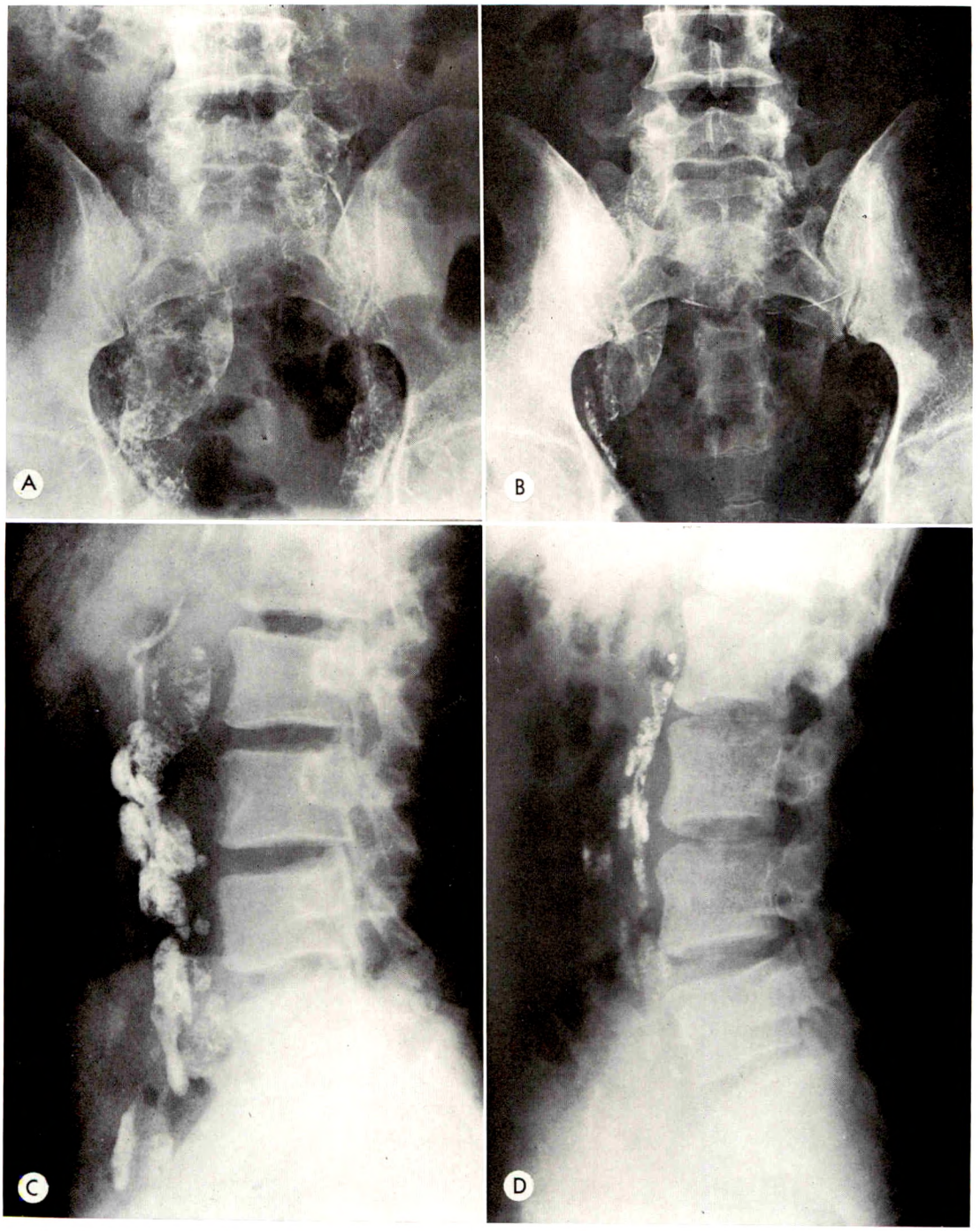


FIG. 8. (*A*) Pretreatment and (*B*) post-treatment roentgenograms of a patient with Hodgkin's granuloma who received radiation therapy. *B* taken two months after start of therapy shows good reduction of lymph node size. Another case of Hodgkin's granuloma (*C*) treated by systemic chemotherapy shows good reduction of retroperitoneal lymph node size following therapy (*D*).

SUMMARY

It has been shown that there are definite clinical applications for lymphangiography. In our experience in the examintaion of 210 patients by this method, we feel that considerable assistance was obtained in the diagnostic and therapeutic management of patients with lymphedema, neoplastic and inflammatory diseases.

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Lymphangiography has been facilitated recently by the use of an automatic pressure regulated injection pump manufactured and donated by the Holter Company, Bridgeport, Pennsylvania.

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IMPROVED TECHNIQUE OF LYMPHANGIOGRAPHY

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LYMPHANGIOGRAPHY is rapidly increasing in popularity. Though its exact role in the investigation of specific problems has not yet been clearly defined, its value as a diagnostic roentgenologic procedure is well recognized. Since it was introduced at M. D. Anderson Hospital and Tumor Institute in November, 1960, lymphangiography has been successfully performed in more than 160 patients.

Production of the lymphangiogram is time-consuming and therefore relatively expensive, and anything which will facilitate the examination and decrease its cost is to be desired. It was this thought that prompted the present investigation of injection technique and injectors.

TECHNIQUE

Lymphangiography was initially performed in the manner described elsewhere[1] with some minor modifications and will be briefly reviewed. The foot is draped and prepared. One-half cc. of an equal mixture of Evans blue dye and 1 per cent procaine hydrochloride is injected intradermally into the dorsum of the web between the first and second toes. Ten minutes later, the skin over the first metatarsal is infiltrated with 1 per cent procaine hydrochloride. A longitudinal 1 cm. incision is made over the first metatarsal, the proximal end overlying the tarsometatarsal joint. A dye-carrying lymphatic is identified by its blue color, isolated, stripped of fat and areolar tissue, and occluded by a small bulldog clamp placed proximally. The lymphatic is then distended by massaging with the thumb from webspace to incision. A 30 inch length of No. 190 polyethylene tubing has been previously fitted with a syringe adaptor on one end and a No. 27 B & D needle on the other. Only rarely is the vessel identified of such a caliber that a smaller needle is required.

The vessel is cannulated with the needle* which is anchored securely in the lymphatic with a single ligature. The adaptor is then connected to the syringe containing 10 cc. of Ethiodol† (an iodinated compound of poppy-seed oil), and the injector (described below) started. Injection is almost invariably bilateral if the lower limbs are used. The same principles apply for injection of the upper limb; modifications are in the amount of contrast medium used (5 cc.), the usual injection of only one upper limb, and the use of a transverse incision just proximal to the anatomic snuffbox.

Roentgenograms of the areas of interest are made immediately following injection, and at 24 hours. Such delayed roentgenograms as may be indicated are also obtained.

CONTRAST MEDIA

Our experience with various other roentgenographic contrast media, as well as stannic oxide suspensions, was unsatisfactory. Ethiodol remains the most useful agent.

INJECTOR

The injectors used in the first 20 cases each consisted of a manually-operated screw arrangement for advancing the plunger of the syringe containing the opaque medium. Manual injection resulted in rupture of lymphatic channels in a high percentage of cases; this is likely attributable to the uneven pressure exerted by too vigorous intermittent advancement of the screw. Lymphatic rupture is often followed by pain, usually minor, in the limb affected. A more serious objection is that the extravasation interferes with proper demonstration of the lymphatic structures.

* This has been likened to performing a barium enema in a gnat, but is really not very difficult.
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The apparatus described was soon replaced by a motor-driven apparatus, which advanced the plunger at an even rate; an injection time of 80 minutes was realized by appropriate gearing of the motor drive and threading of the advancing screw. As a consequence, rupture of the lymphatics almost never occurred. These injectors were used in 98 cases. However, the increasing popularity of the procedure indicated that several patients would have to be examined simultaneously, and further injectors would be required. To decrease expense and simplify the procedure, the possibility of using a gravity feed was investigated.

Measurements in the system during the injection indicated pressures in the neighborhood of 1,100 mm. Hg (21 pounds per square inch) in the syringe. This surprisingly high pressure is largely dissipated in overcoming the resistance of the long length (30 inches) of small bore polyethylene tubing and is required to expel the ethiodol from the needle at the desired rate of 0.12 cc. per minute. It was found that a weight of 9.5 pounds must be placed on the plunger to achieve this flow rate when a vertically positioned syringe is used. The syringe employed is a 20 cc. Ideal with an internal caliber of 2.0 cm.

A lucite frame that would support the two syringes in vertical position was constructed. The exact dimensions are unimportant, and will not be reproduced. A lead cap weighing 9.5 pounds was machined to fit over each plunger. This weight is so shaped that its center of gravity is below the midpoint of the area of support and equilibrium is therefore stable. Two views of the apparatus in use are shown in Figure 1, *A* and *B*.

Before injection is started, the frame is levelled so that the axes of the syringes are vertically aligned, though minor deviations from the vertical do not appear to be critical. The plunger and barrel of each syringe are lubricated with the opaque medium. During the injection, the technician rotates the barrel in the plunger by rotating the weight at intervals of approximately 15 minutes. All these measures are designed to preclude sticking of the plunger in the barrel. This has been used in 35 patients (67 injections) without difficulty.

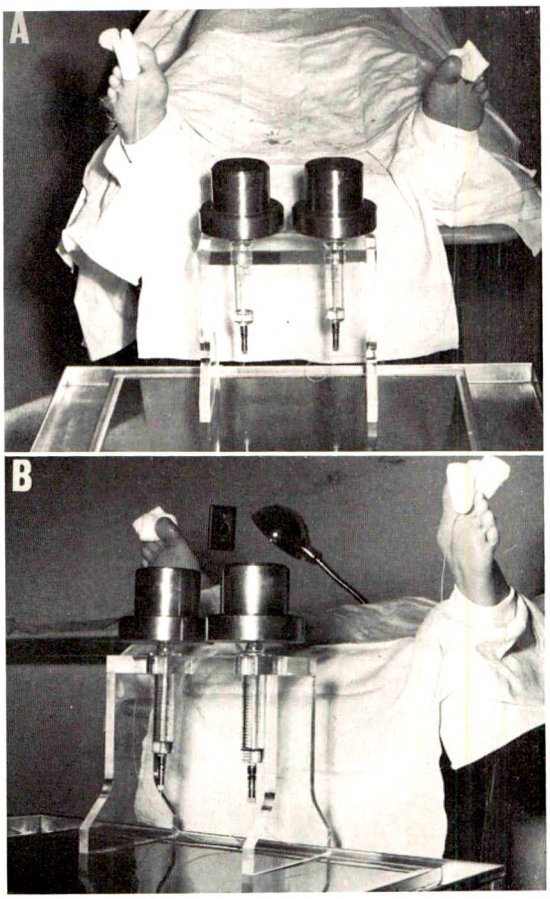


Fig. 1. (*A* and *B*) Two views of the injector presently in use.

SUMMARY

The technique of lymphangiographic injection is discussed with special attention to the injectors used.

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ANGIOARCHITECTURE OF THE SKIN*

MICROANGIOGRAPHIC STUDIES

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THE skin performs two important functions; *i.e.*, it serves as a thermoregulator and as an organ of blood storage; these functions depend directly on the nervous regulation by the diencephalic nuclei.

The essential elements of thermoregulation are the arteriovenous anastomoses; these are short vessels with well-defined structural characteristics of the wall allowing wide and rapid changes of caliber which alter the hemodynamic conditions so that a larger quantity of blood may pass through them per unit of time. Such anastomoses are present in the entire body, as has been shown by Clara,[1] Grant and Bland,[7,8] and Goodall.[6] There may be more in certain zones, such as in the skin where their caliber exceeds 50 microns. In the skin of the hands and feet their number may range between 250 and 600 per square centimeter. Indeed, only large intervascular bridges, *i.e.*, short and wide canals such as the arteriovenous anastomoses, might allow a rapid flow of blood greater than would be possible by a simple enlargement of the nutritional system, the resistance of which would be much higher.

In order to fulfill its functions of blood storage, the skin needs a wide vascular bed of large storage capacity, such as the subpapillary venous plexuses. As a storage organ, the skin also plays an important part in hemometakinesia, *i.e.*, the transference of blood from one part of the body to another. Often, the conveyance of blood must take place rapidly when important organs such as the brain and the heart are in need but, at other times, only small blood transferences occur induced by small stimuli, sometimes of emotional origin.

There is need for a revision of our knowledge of the physiology and the physiopathology of the microcirculation. Microangiography is particularly suitable for the study of structures which constitute a link between the vessels visible to the naked eye and the finer elements such as the capillaries. By advancing the concept of macroreticulum and microreticulum, Saunders and his collaborators[12] ascribe to the intermediary vascular structures the most important functions in the microcirculation.

MORPHOLOGY

The complexity of the functions of the skin demands increased knowledge of the morphology of its vascular network. This paper presents our initial findings on the angioarchitecture, both arterial and venous, of human skin. It might be worthwhile, however, to begin by reviewing Petersen's[10] description of this angioarchitecture. The vessels for the skin derive from the muscular arterial branches. At the junction of the dermis with the hypodermis, there is the arterial cutaneous plexus described by Spalteholz,[13] the venous plexus and the coils of sweat glands. This zone situated between the dermis and the hypodermis is thus also called vasculoglandular. From the arterial cutaneous plexus derive the so-called "candelabrum" arteries which are arranged horizontally in the overlying dermis. These are long arteries of small caliber which, after following an arciform course, return to the cutaneous network. From the "candelabrum" arteries emerge the arterioles, whose function is to irrigate the adipose lobes and the sweat glands, which are also supplied by the cutaneous network. In the compact layer of the corium there are arciform anastomoses connecting the "candelabrum" arteries which, owing to their great number, give rise to

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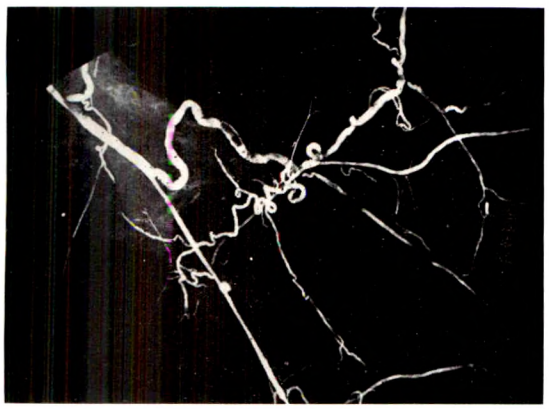


Fig. 1. Arterial system of the skin of the upper eyelid. (Reproduced by permission of *Annales de Radiologie*, No. 7, 1961.)

Spalteholz's subpapillary arterial plexus. From this plexus, vessels emerge which divide into branches as far as the papillae.

On the palms of the hands and the soles of the feet, as well as on the ungual limbus, there are fine arteries which give rise to the capillaries. These supply blood to the capillary venous network. Another venous network is formed by veins of large caliber which correspond, in the arterial system, to the first ramifications of the "candelabrum" arteries.

TECHNIQUE

We used necropsy specimens of individuals over 60 years of age.

For study of the palpebrae and scalp, we chose to inject the common carotid artery. After tying the distal end and performing a perfusion with normal saline solution, micropaque aqueous solution at 50 per cent was injected. The mean size of the barium particles was 0.1 μ to 0.2 μ which allows easy filling of the finest capillary structures. About 30 cc. injected by manual pressure is enough for a good opacification, which can be verified by the white coloration of the tegument.

The contrast medium was injected into the radial artery for study of the vessels of the dorsum of the hand, and into the femoral artery for those of the dorsum of the foot.

After the injection was completed, skin specimens of about 2 cm. by 2 cm. were prepared by dissecting them from the subcutaneous cellular tissue and from the fat. The specimens were then fixed on a small wood board with four drawing pins, but without being unduly stretched, and were submerged in a 10 per cent formol solution for 48 hours for fixation purposes.

For roentgenographic exposure we employed a Bucky therapy apparatus with a Machlett tube AEG 50 having a 1 mm. focus and beryllium window. Kodak maximum resolution plates, with a resolution power of 1,000 lines, were used.

The roentgenographic factors were:

	kv.	ma.	Focus Film Distance (cm.)	Exposure (min.)
Palpebra	20	10	19	2
Dorsum of the foot	20	10	19	2
Dorsum of the hand	20	10	19	2
Pulp of the finger	25	10	19	2
Scalp	25	10	19	3

FINDINGS

ARTERIAL SYSTEM

Our microangiographic studies seem to entirely confirm the classic description of the angioarchitecture of the skin. They further reveal characteristic arteriolar patterns in some regions of the human body which have an anatomic and, perhaps, also a functional basis. In the eyelid, the dorsum of the hand and the dorsum of the foot, where the skin is rather distensible, it was found that the arteriolar pattern is strikingly analogous. A description of this arteriolar pattern follows.

Eyelid. Figure 1 is of the skin of the upper eyelid and shows a vein (left) with a rectilinear branch, and another of larger caliber, which is slightly tortuous. At right angles to the venous vessel is a tendril-like arteriole which is perpendicular to the palpebral commissure; *i.e.*, it turns in the same direction in which the palpebra distends. The collateral branches are rectilinear and

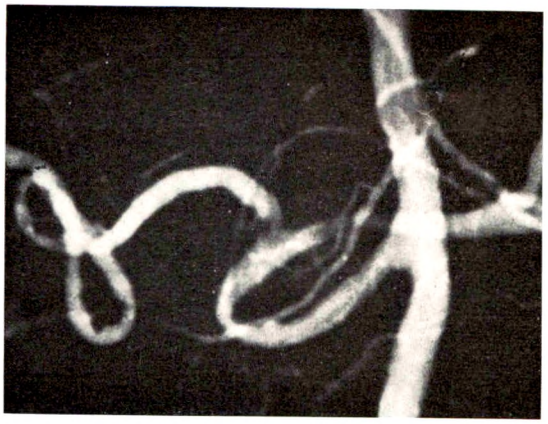


FIG. 2. Microscopic view of microangiogram of the arterial system of the skin of the dorsum of the hand, showing coil-like vascular pattern.

arranged in wide arcades. The existence of a similar vascular configuration in the striated muscles has already been shown by Saunders *et al.*[12] and Veiga Pires.[14,15] Freitas de Sousa,[5] after injecting a tenotomized muscle and its antagonist, concluded that, for reasons related to the physiologic mechanisms, the muscular arterioles and capillaries can show in varying degree a rather tortuous course.

Dorsum of the Hand. On the dorsum of hand, *i.e.*, at the level of the metacarpophalangeal joints, some coil-like vascular patterns were frequently found. Such an arrangement cannot be the result of any technical artefact, since we fixed the specimen in such a way as to avoid retraction caused by the formol. Also, the same microangiogram shows other vessels which are straight. Looking at the microangiogram under the microscope, it is possible to follow the tortuosities of the vessels and one gains an impression of depth. Figure 2 shows clearly this particular configuration. A collateral vessel gives origin to two branches, one of them forming a double loop coil and the other crossing the original vessel perpendicularly. Vessels of much smaller caliber are seen to arise at various sites.

Dorsum of the Foot. In a cross-section of the skin of the dorsum of the foot, the arrangement of the cutaneous vessels is well demonstrated to be in agreement with Petersen's[10] description (Fig. 3). In the lower part we see 4 afferent vessels belonging to the subcutaneous network and assuming a more or less parallel course. It is not possible to demarcate the various skin layers, but the caliber and configuration of the vessels enabled us to barely distinguish the arterial plexus. From this plexus originate the "candelabrum" arteries which anastomose with one another, forming a compact network.

Scalp. In the skin of the scalp the architectural arrangement of the arterial vessels is quite different. We found slightly sinuous vessels of relatively large caliber connected by anastomoses of small caliber, forming a large-meshed reticulum. There are, as it were, rectangular and polyhedral areas towards the center of which very fine branches are directed. It is possible that such a vascular arrangement may explain the small islands of coloration found by some researchers[4] who used injections of India ink.

The arterioles of the scalp seen in cross-section (Fig. 4) show in the terminal portion small hooks resembling vine tendrils. We theorize that such structures correspond to terminal stems of the "candelabrum" arteries arranged circularly around the roots of the hairs.

Pulp of the Finger. In the skin of the pulp of the finger it has not been possible to isolate the arterial system from the circula-

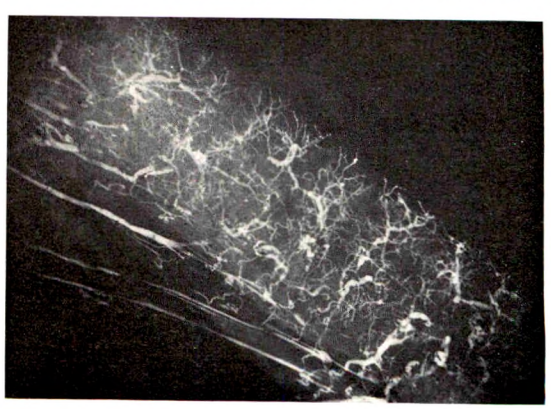


FIG. 3. Arterial system of the skin of the dorsum of the foot.

tion, since the contrast material injected into the arterial system passed rapidly into the venous structures, probably due to the large number of arteriovenous anastomoses. The result, therefore, was the superimposition of the arterial and venous systems; however, this, in itself, is of some interest.

The microangiogram (Fig. 5) cannot be confused with that of any other part because the arterial and the venous vessels are arranged according to the papillary mounds and correspond exactly to the fingerprint. However, the cross-sections demonstrate more clearly the subpapillary capillary network. Figure 6 shows the capillary loops which derive from the distal vessels of the "candelabrum" arteries. As the papillae are more prominent here than in any other cutaneous areas and are regularly arranged, we may more easily observe the characteristic angioarchitecture.

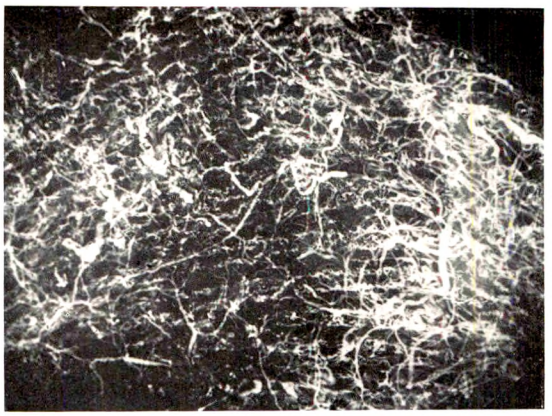


Fig. 5. Vascular pattern of the skin of the pulp of the finger.

VENOUS SYSTEM

Eyelid. The architecture of the fine structures of the venous network in the eyelid appeared to be a most typical one (Fig. 7). Longitudinal straight vessels, of more or less uniform caliber, were found converging in an orderly manner towards the edge of the palpebra. (It should be noted that the microangiograms were obtained with the skin distended as described above under "Technique.") Among the longitudinal microcollectors there are tortuous anastomoses of uniform caliber and also arranged with a remarkable regularity. The whole picture is that of a reticulum of wide and regular mesh. However, inside each space there are other vessels of much smaller caliber also forming a reticulum. It thus appears logical to include the fine venous circulation in the concept of macroreticulum and microreticulum described by Saunders *et al.*[12] for the arteriocirculation of the muscle.

With greater distention of the specimen, the vascular structures become rectilinear. With greater magnification the large and the small venous anastomoses can be seen more clearly. Figure 8 shows a longitudinal canal (left) and includes two complete meshes and part of another one from which emerge vessels of small caliber, possibly



Fig. 4. Arterial system of the scalp.

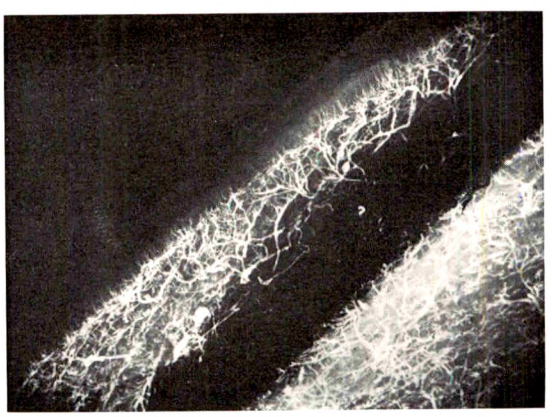


Fig. 6. Subpapillary capillary network of the skin of the pulp of the finger.

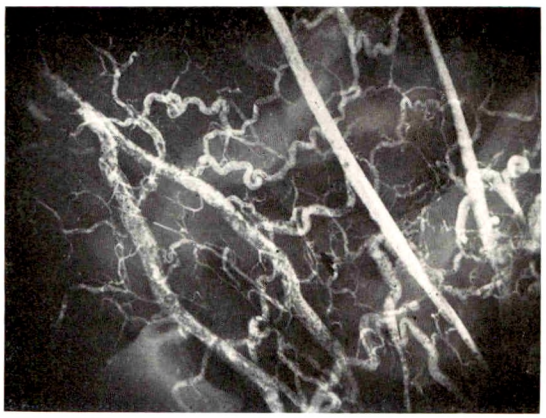


FIG. 7. Venous system of the skin of the upper eyelid.

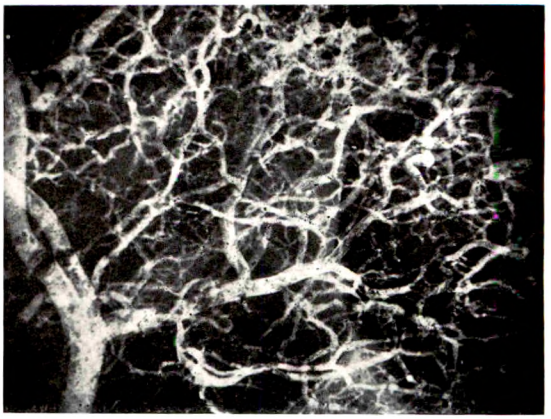


FIG. 9. Venous system of the skin of the ear.

postcapillary venules. It is in the partly shown mesh that the small anastomoses are seen to form a fine reticulum to which the capillary venous trunks most probably contribute.

Ear. The venous system of the skin of the ear is entirely different and is characterized by an obvious difference in the caliber of the vessels and by the density of the network. Figure 9 demonstrates a dense entanglement of venules which divide irregularly and also cross each other in an irregular way. Considered individually, the small branches are short and maintain practically the same caliber through their whole course. When tortuosities are present, they appear slightly marked. In the middle of the network of small vessels a much thicker vessel stands out, which, on dividing, reduces slightly in caliber. The caliber of the further branches diminishes suddenly. Sometimes, the main branch trifurcates, showing on the whole a typical shrub appearance. Here again there is an arrangement of the venous network in macroreticulum and microreticulum.

Dorsum of the Foot. In the skin of the dorsum of the foot, there is a venular architecture with well-marked characteristics peculiar to the lower limb because of the presence of valves.

By injecting the contrast medium retrogradely into the thicker venules, where the caliber is 15 times smaller than that shown in Figure 10, the venular system resembles a pruned tree. From the two bifurcated branches derive short segments with sharp terminations which seem to be amputated

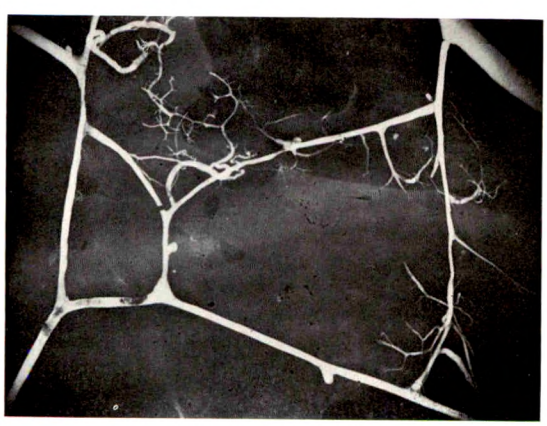


FIG. 8. Magnification of the venous anastomoses of the skin of the upper eyelid.

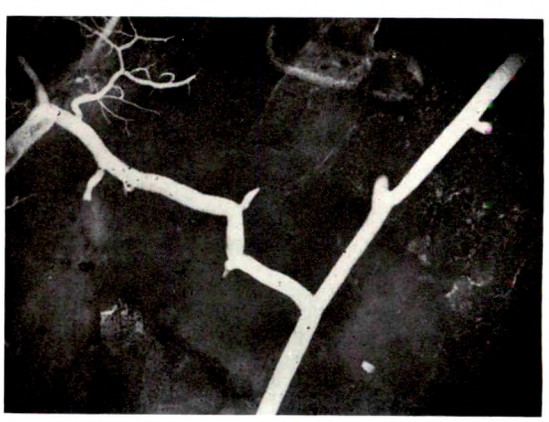


FIG. 10. Venous system of the skin of the dorsum of the foot. Pruned tree appearance in the presence of competent valves.

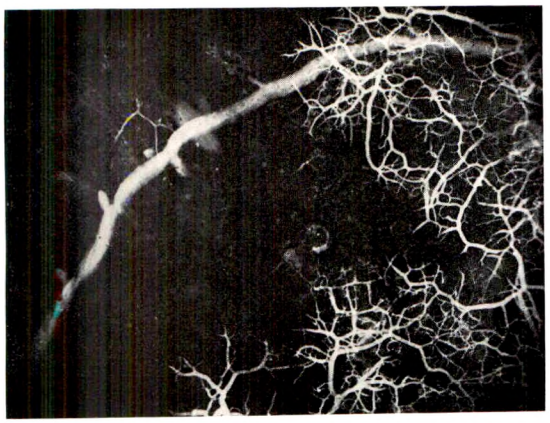


Fig. 11. Venous system of the skin of the dorsum of the foot. Insufficiency of 2 valves permitted filling of a large number of tributary vessels.

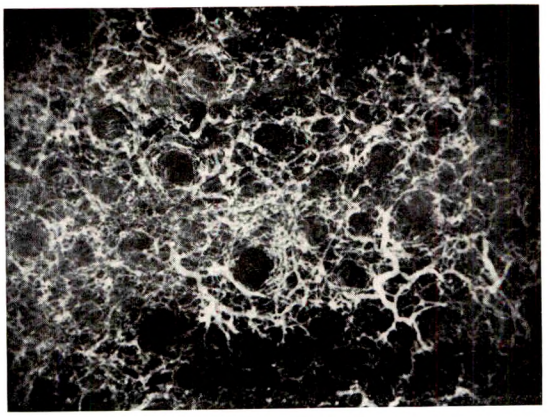


Fig. 12. Venous system of the skin of the nose. Note bird nest appearance.

obliquely or transversely; these present at more or less fixed distances and are orientated alternately inwards and outwards. Such a pattern obviously suggests a mechanical barrier to the retrograde flow of the injected medium, *i.e.*, a valvular system.

The larger vessels are long and sometimes arciform. In Figure 11 in a number of vessels the valvular filling was sufficient to reproduce the pattern seen in Figure 10. Distally, 2 valves gave way to the pressure of the injection or were already incompetent and allowed filling of a large number of tributary vessels. Their caliber is greatly reduced and they are not regularly arranged. In the middle of the reticulum formed by short vessels without great differences in caliber, some longer vessels forming bridges between valvulated passages are easily seen.

Nose. In the skin of the dorsum of the nose the venous structures have an extremely curious arrangement. In the middle of the reticula, there are blank round zones of about the same size and separated by equal distances, around which the vessels are circularly arranged giving the appearance of bird nests (Fig. 12). We cannot offer an accurate interpretation of these apparently nonvascular zones. Nevertheless, pending further research, we postulate that such a pattern is perhaps connected with Horstmann's[9] rosettes and is related to the follicle of the hair and the ducts of the sebaceous glands.

SUMMARY

The microangiographic method was used for the study of the angioarchitecture, both arterial and venous, of the human skin. The technique is briefly discussed.

The existence of a typical angioarchitectural pattern in the more distensible zones of the skin, such as the palpebra and dorsum of the hand, is demonstrated. On the pulp of the fingers it was found that the vascular network reproduces exactly the pattern of the fingerprints. In cross-sections of the same area the "candelabrum" arteries and the arteriovenular system of the dermal papillae are clearly individualized.

Very characteristic arrangements were also found in the venous network. In the palpebra, a system of vessels was noted which was arranged perpendicularly to the commissure and formed extremely long arches. A venous network of wide mesh was observed in the skin of the scalp and a shrub-like venous pattern in that of the ear. The venous network in the skin of the dorsum of the nose with a configuration resembling that of bird nests is possibly related to Horstmann's rosettes.

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VOL. 88, NO. 1

ROENTGEN DIAGNOSIS OF AIR EMBOLISM DUE TO CRIMINAL ABORTION*

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THE purpose of this communication is to report the use of roentgenography as a diagnostic method in air embolism resulting from the performance of criminal abortions. Venous air embolism doubtless occurs much more frequently as a complication of criminal abortions than the number of instances recorded in the literature would indicate because some of these unfortunate accidents may not be discovered and most of them are never reported. The pathologist, medical examiner and coroner welcome any aid in the establishment of this important diagnosis, particularly a method which is applicable prior to the performance of an autopsy. Some of the procedures formerly utilized were the opening of the heart with the body immersed in water and the insertion of a needle into the cardiac chambers, the contained air being withdrawn into a syringe or bottle filled with water. These methods are cumbersome and subject to error because of technical difficulties. With the use of roentgenography, on the contrary, the presence of air can be clearly demonstrated in the chambers of the heart, the veins, and the soft tissues of the pelvis without disturbance of the anatomic relations of the body structures. The roentgen manifestations are graphic, definite and reliable. We have found roentgenography a means of establishing the diagnosis and of confirming the clinical findings or impressions in suspicious cases. Roentgen studies supply incontrovertible evidence, often in a simpler fashion than can be obtained otherwise. It has the added advantages of not interfering with routine or special postmortem studies. Roentgenography is easily carried out as the examination can be performed with the equipment available in every x-ray department and requires neither special apparatus nor complicated techniques. The studies can be repeated if necessary and are useful in the confirmation of evidence afforded by other diagnostic procedures, which may be significant from the medicolegal point of view. Another important factor is that cases in which the possibility of an abortion has not come into consideration because of lack of an adequate history and/or the absence of suspicious details may be uncovered as the result of routine roentgen examination.

CLINICAL AND PATHOLOGIC CONSIDERATIONS

Venous air embolism, also known as gas embolism or pulmonary venous embolism, results from the introduction of moderate or large amounts of air into the venous circulation. There is cyanosis and immediate death due to anoxia. The event is a dramatic one and attempts at resuscitation are futile as a rule. It may occur in surgical operations or other trauma involving the larger veins; in association with pregnancy as a rare complication of delivery or postpartum surgical sterilization; as the result of the assumption of the knee-chest position in the performance of exercises during the puerperium; as a complication of pneumothorax and pneumoperitoneum; while douching or insufflating powder into the uterus; in the administration of fluid intravenously for therapeutic or diagnostic purposes; as the result of direct injection of air into the circulation for purposes of euthanasia; and in the performance of a criminal abortion. A common procedure in performing an abortion is the insertion of a rubber tube or nozzle into the uterus. Air under pressure may enter along with the fluid. Detachment of the placenta, injury to the vessels, or a combination of both favors

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the passage of air into the uterine sinuses. This is particularly apt to occur in the case of the pregnant uterus because of the increased vascularity and the friability of the vessels. The air passes into the pelvic, iliac, abdominal, and hepatic veins and also into the inferior vena cava, the right cardiac chambers, and the pulmonary arteries. It enters the superior vena cava and reaches the neck and head by retrograde movement, and is found in the jugular, the cerebral, and the large veins at the base of the brain.

It is essential that the roentgen studies be performed in the period immediately after death as gas may form in the blood vessels and other areas of the body within a matter of hours by the action of gas-producing organisms. It must also be borne in mind that certain postmortem procedures may cause gas to pass into the blood vessels and the heart, and this possibility must be excluded before the diagnosis of air embolism is made.

ROENTGEN MANIFESTATIONS

In postmortem roentgenography, the examination is commonly made with the body recumbent since other positions are difficult to maintain. To establish the diagnosis of air embolism definitively, however, roentgen studies with the body supine or prone must be supplemented by studies (1) in the upright position and (2) lateral decubitus views utilizing a beam of roentgen rays directed horizontally. In performing these studies it is advisable to support the body by straps or other suitable device; the cassette containing the roentgen ray film should also be held in place mechanically. If the service of an assistant is utilized for these purposes, adequate protection must be provided by means of shields, leaded gloves and aprons. A collimating cylinder is helpful in affording additional protection to personnel and in improving the detail of the roentgenograms. The roentgen manifestations comprise the following: (1) air in the right side of the heart and the pulmonary artery with an air-fluid level in the right ventricle, the level remaining horizontal regardless of the position of the patient; (2) visualization of the veins of the torso, neck and head due to the presence of air within these structures; and (3) air shadows in the soft tissues of the pelvis and lower abdomen (Fig. 1, *A*, *B* and *C* and Fig. 2, *A*, *B* and *C*).

THE CHEST

With the body recumbent, the sagittal roentgenogram shows the lungs clear or with varying degrees of increased density due to postmortem changes or associated pulmonary pathology. There is increased radiance in the region of the heart shadow, this radiant area occupying the site of a portion of the cardiac silhouette. Band-like areas of increased radiance in the region of the larger vessels of the upper portion of the chest, the neck, and the liver region are manifestations of gas in the veins. With the body in the standing or sitting position, the right ventricle presents an air-fluid level, the lower segment of the right ventricle being of uniform density because of the blood contained within it. The upper portion of the right ventricle, the right auricle, the main pulmonary artery and its principal branches, the superior vena cava and the veins of the upper chest are outlined by air. Lateral decubitus views utilizing a horizontal roentgen ray beam show an air-fluid level in the right ventricle with air in the venous structures as described above; this portion of the examination is best performed with the body lying on the left side and the right side uppermost.

THE PELVIS AND ABDOMEN

Studies of the pelvis and abdomen are made with the body recumbent. A Bucky grid is essential. The exposure factors are similar to those utilized in the examination of the urinary tract or lumbar spine. The pelvis shows multiple, linear and band-like areas of increased radiance extending longitudinally or obliquely. These occupy the lateral aspects of the pelvis and may extend into the lower abdomen. In the abdomen,

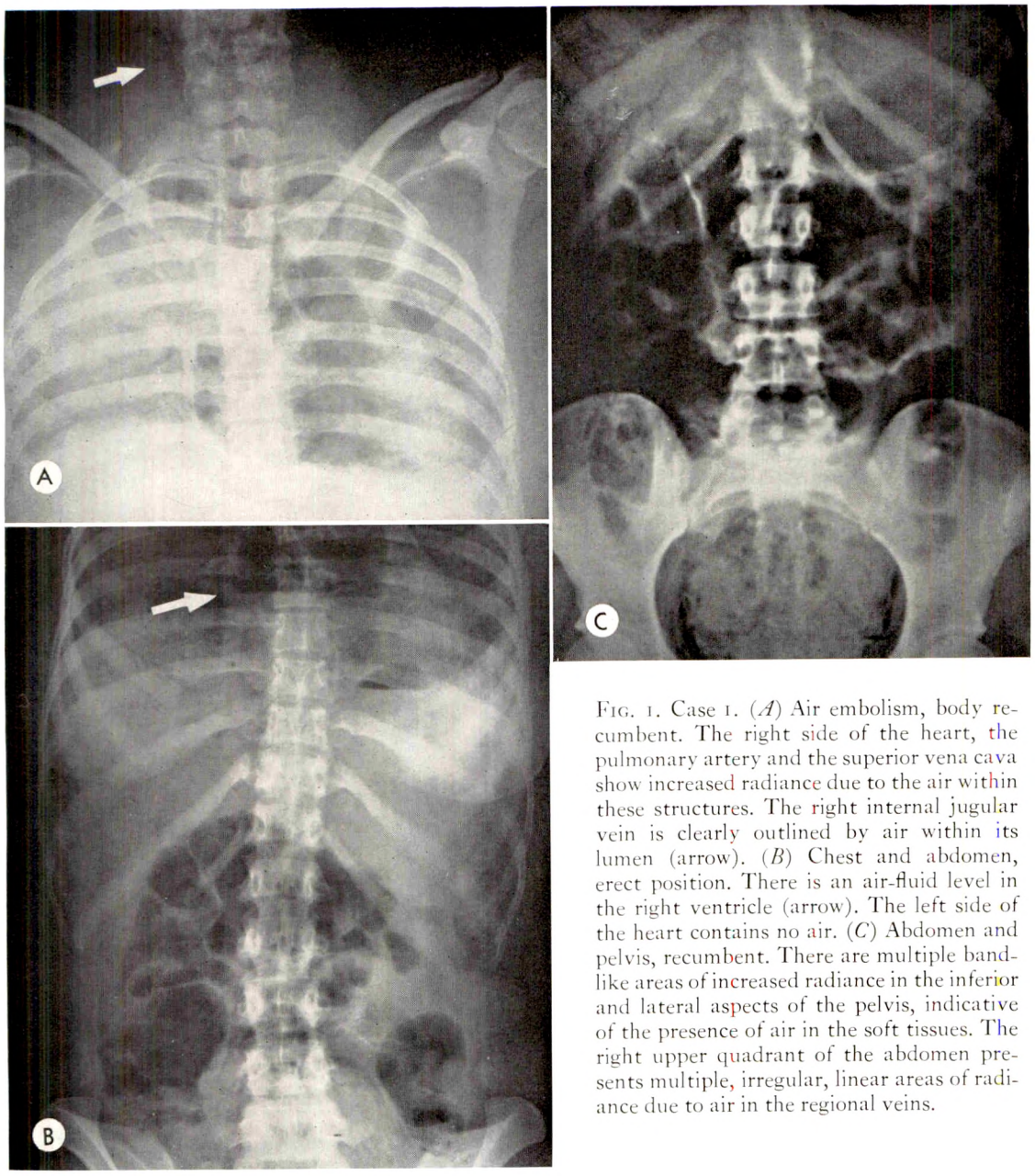


Fig. 1. Case 1. (*A*) Air embolism, body recumbent. The right side of the heart, the pulmonary artery and the superior vena cava show increased radiance due to the air within these structures. The right internal jugular vein is clearly outlined by air within its lumen (arrow). (*B*) Chest and abdomen, erect position. There is an air-fluid level in the right ventricle (arrow). The left side of the heart contains no air. (*C*) Abdomen and pelvis, recumbent. There are multiple band-like areas of increased radiance in the inferior and lateral aspects of the pelvis, indicative of the presence of air in the soft tissues. The right upper quadrant of the abdomen presents multiple, irregular, linear areas of radiance due to air in the regional veins.

the air-filled veins produce a network of narrow bands of increased radiance, the changes being most clearly visualized in the right upper quadrant in the region of the liver.

NECK AND HEAD

The venous structures of the neck and skull may be outlined by air contained within them. Many of the veins can be identified by their anatomic location.

DIFFERENTIAL DIAGNOSIS

The principal condition which must be considered in differential diagnosis is hydropneumopericardium. Air may gain entrance into the pericardial sac during tapping of the pericardium or pleura: secondary to

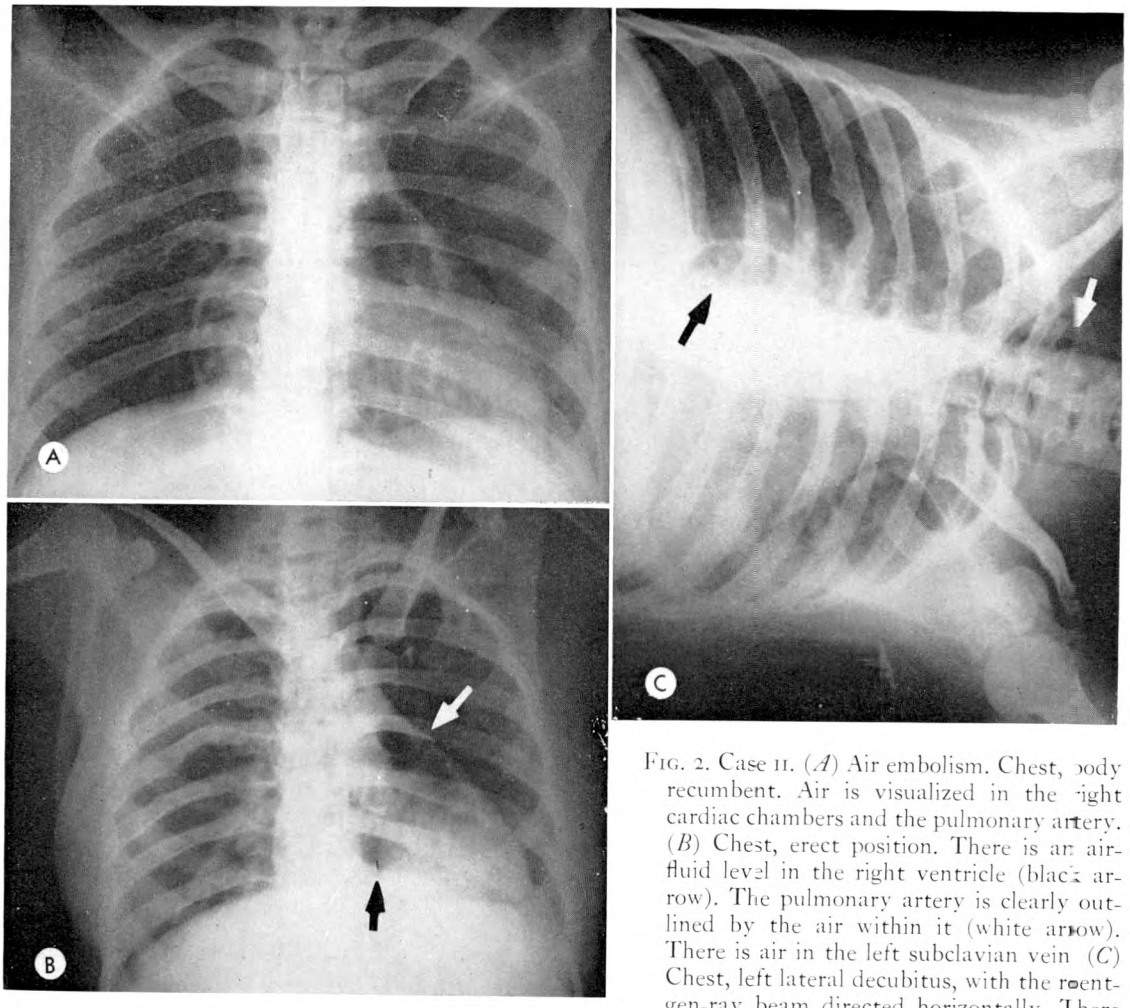


FIG. 2. Case II. (*A*) Air embolism. Chest, body recumbent. Air is visualized in the right cardiac chambers and the pulmonary artery. (*B*) Chest, erect position. There is an air-fluid level in the right ventricle (black arrow). The pulmonary artery is clearly outlined by the air within it (white arrow). There is air in the left subclavian vein (*C*) Chest, left lateral decubitus, with the roentgen-ray beam directed horizontally. There is an air-fluid level in the right ventricle (black arrow). The right internal jugular vein is clearly outlined by gas (white arrow).

malignant disease of the esophagus, lung, mediastinum and stomach; and after trauma to the chest. The roentgenographic differentiation from air embolism is based on (1) the visualization of the pericardium as a linear band of density separated from the heart by the increased radiance caused by the air in the pericardial sac and (2) the different type of air-fluid level. The air-fluid level in hydropneumopericardium is wider than in air embolism, extending across the entire width of the heart shadow rather than being limited to the right ventricle. In the erect lateral views, the air is visualized in the retrosternal region, rising to the upper portion of the pericardial sac. The right lateral decubitus study is of particular value in demonstrating that the air and fluid are in the pericardial sac rather than in the right chambers of the heart. Also, air is not visualized in the venous structures of the pelvis, abdomen, chest, neck, and head in hydropneumopericardium.

In pneumomediastinum, there are crescentic or linear streaks of increased radiance overlying the heart; these extend to either side of the midline in the region of the superior and middle portions of the mediastinum, along the margins of the great vessels of the root of the neck, and into the pericardium. Dissecting aneurysm of the

aorta or rupture of an aortic aneurysm may result in the rapid accumulation of fluid in the mediastinum. Fluid levels do not occur in any of these conditions and there is little likelihood of confusion in diagnosis. Cardiospasm with large amounts of fluid and air in the markedly dilated esophagus is differentiated by the fact that the gas shadows extend beyond the borders of the heart and rise into the neck.

In the case of the gas shadows which are visualized on the roentgenogram of the pelvis after criminal abortion, the principal conditions to be considered in differential diagnosis are emphysematous cystitis and the changes which may result from the presence of gas forming organisms in the pelvic soft tissues. Emphysematous cystitis is characterized by curvilinear areas of increased radiance arranged in concentric fashion in the region of the margin of the bladder. The changes which result from the action of gas forming bacilli in the pelvic soft tissues are distributed irregularly, tend to be more variable in size and may involve only a portion of the pelvis or the entire pelvis. In neither of these is there air in the heart or the venous structures.

As with other rare and unusual conditions, the diagnosis will be made roentgenographically only if the manifestations are known and clearly understood. Since the establishment of the diagnosis of venous air embolism is a matter of great importance from the medicolegal point of view, the possibility of the condition must be borne in mind and carefully sought for by appropriate roentgen studies in all suspicious cases.

SUMMARY AND CONCLUSIONS

In the past roentgenography has found only a limited field of usefulness in postmortem studies. Its principal applications have been: (1) the identification of bodies; (2) the detection and localization of bullets, missiles, and other opaque foreign bodies; (3) the correlation of clinical and pathologic findings; and (4) teaching and research. We have found roentgenography a reliable and relatively simple method of establishing the diagnosis of venous air embolism. It may serve as an important aid in determining the cause of death in certain cases of criminal abortion. The demonstration of the presence of air in the right cardiac chambers, the venous system and the pelvic tissues does not require special roentgenographic apparatus nor complicated roentgen techniques. The method has the added advantage that it may call attention to the condition in cases in which the possibility was not considered because of lack of a history or absence of details which might arouse suspicion in the mind of the examiner.

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CALCIFICATION, OSSIFICATION AND CARTILAGE FORMATION IN THE KIDNEY*

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THERE is relatively little information in the urologic and roentgenologic literature concerning the occurrence of calcification, ossification and cartilage formation in nontuberculous diseases of the kidney. Our interest in this subject was stimulated by encountering several unusual cases which exhibited calcium shadows in the renal region and which presented problems in diagnosis.

I. *Calcification.* Calcifications have been observed in a wide variety of pathologic lesions of the kidney. From a practical standpoint, calcification of the kidney may be divided into two main types: metastatic and nonmetastatic. In the metastatic type the abnormal deposition of calcium salts in the kidney is associated with their increase in the blood, and presumably occurs in healthy kidney tissue as a result of faulty renal excretion of these salts. The nonmetastatic type of calcification occurs in renal tissue which is the site of a localized or diffuse lesion, like fatty degeneration, hyalinization, inflammation, suppuration or necrosis resulting from a toxic, circulatory, inflammatory or metabolic process and predisposing to the deposition of calcium salts. The nonmetastatic type of calcification may be subdivided into the following subgroups: (1) dystrophic calcification, (2) heterotopic calcification, (3) cartilage formation, (4) arterial calcification, (5) nephrolithiasis and (6) nephrocalcinosis. Discussion will be limited to the first four groups. The last two groups represent distinctly different forms of calcification which have been the subject of a report presented elsewhere by one of the authors (B.S.A.). The important factor in the nonmetastatic type of calcification is the presence of a pathologic lesion in the kidney. Hypercalcemia is not essential, but the degree of calcification is usually more marked in cases accompanied by a transient or permanent hypercalcemia. Dystrophic calcification is the most common type of nonmetastatic calcification, as any damaged or dead tissue may become infiltrated with calcium salts.

II. *Ossification (osseous metaplasia, heterotopic bone formation).* Ossification of the kidney is a rare type of nonmetastatic calcification. The close interrelationship between calcification and ossification from a physiologic standpoint is well recognized. The pathologic aspects of ossification of the kidney have been confirmed by numerous experimental studies of heterotopic bone formation in the urinary tract. While it is generally agreed that the formation of heterotopic bone in the kidney is dependent upon a metaplasia of the connective tissue, the exact mechanism responsible for the deposition of calcium salts in the affected area is not yet clear. Probably more than one factor is involved, like the stimulating influence of infection or irritation, an alteration of pH of the tissue fluid, the availability of calcium and phosphorus in the urine and blood and the action of the phosphatase enzyme. The neoplastic theory has been advanced to explain ossification in the rare cases of mixed tumor dermoid cysts and osteosarcoma. These tumors are of mesoblastic origin and present unusual embryonal features.

III. *Cartilage Formation.* Cartilage formation is never encountered in the normal kidney and has been rarely observed in

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a relatively few renal diseases, like polycystic disease or Wilms' tumor.

We have adopted the classification of destructive or degenerative lesions of the kidney associated with dystrophic calcification which is shown in Table I. It is the basis on which we have organized the following exposition of this subject.

A. Acute and Chronic Infections

Pyelonephritis. Calcification is practically never associated with acute pyelonephritis, but it may be found with chronic infection of the kidney, such as suppurative or cicatrizing pyelonephritis, localized or diffuse abscess formation, carbuncle, pyonephrosis, etc. Usually calcium deposits are not discernible roentgenologically (Fig. 1) but they can be readily identified on histologic examination. Calcification has also been occasionally described in cases of necrotizing papillitis, which is a severe form of acute pyelonephritis. Lusted *et al.*[16] reported papillary calcification demonstrated on laminagrams in a case of necrotizing papillitis. Diffuse calcifications may occur in one or more pyramids and microlithiasis in and around the basement membrane of the collecting tubules in cases of calyceal calculi due to urea splitting organisms. In more than 40 per cent of 60 cases of calyceal calculi treated by partial nephrectomy, this finding was noted by one of the authors (B.S.A.). After chronic suppurative pyelonephritis, calcifications in the dense cicatricial tissue in the cortical areas may occur.

Abscess. In cases of chronic abscess formation secondary to urinary stasis and infection, it is not uncommon to find cavities containing thick pus and calcareous particles adherent to the lining of the walls, but usually they cannot be seen roentgenographically.

Bilateral Cortical Necrosis. This disease runs a rapidly fatal course and, consequently, the opportunity to study these cases is seldom afforded. With the introduction of the artificial kidney, it is now possible to sustain life for longer periods of time, and occasionally to obtain a cure. The

Table I

Destructive or Degenerative Lesions of the Kidney Associated with Dystrophic Calcification

- A. Acute and Chronic Infections
  - 1. Pyelonephritis
  - 2. Abscess (single or multiple), carbuncle
  - 3. Bilateral cortical necrosis
  - 4. Infection of peripelvic and peri- and paranephritic tissues
  - 5. Tuberculosis
  - 6. Bilharziasis
- B. Obstructive Lesions
  - 1. Hydronephrosis
  - 2. Pyonephrosis
- C. Tumors of the Kidney
  - 1. Cystic lesions
    - a. Solitary serous cyst
    - b. Hemorrhagic cyst
    - c. Multilocular cyst
    - d. Peripelvic or parapelvic cyst
    - e. Polycystic disease
    - f. Congenital unilateral multicystic kidney
    - g. Echinococcus cyst
  - 2. Benign tumors
    - a. Adenoma
    - b. Fibroma
    - c. Hamartoma
    - d. Miscellaneous
  - 3. Malignant tumors
    - a. Adenocarcinoma (including hypernephroma)
    - b. Papillary cyst adenocarcinoma
    - c. Papillary carcinoma of renal pelvis
    - d. Squamous cell carcinoma of renal pelvis
    - e. Osteosarcoma (primary and secondary)
    - f. Osteoma (pararenal teratoma, dermoid cyst)
    - g. Miscellaneous
- D. Traumatic Injuries
  - 1. Intrarenal
    - a. Calcification
    - b. Calcified cysts
  - 2. Extrarenal
    - a. Perirenal hematoma
    - b. Perirenal cyst
    - c. Pseudohydronephrotic cyst
- E. Vascular Disease
  - 1. Arterial calcification and/or aneurysm of renal artery
  - 2. Infarct
  - 3. Vascular tumors (endothelioma)
- F. Lithiasis
  - 1. Calcification and ossification associated with renal calculi
  - 2. Calcareous incrustation of pelvic and calyceal lining

diagnosis is established by biopsy and roentgen studies. At the onset, the pathologic symptoms are those of an acute tubular nephritis, such as tubular or lower nephron necrosis. Later, renal insufficiency ensues with progressive oliguria and anuria, and the findings indicate a bilateral cortical necrosis. In the early stage, the outline of the kidney is larger than normal, but later it becomes normal or, in a few cases, smaller than normal; subsequently, calcifications may appear.

Infection of Peripelvic, and Peri- and Paranephritic Tissues. The presence of calcification is rarely observed following an acute inflammatory or suppurative process. Occasionally, marked sclerosis and fibrosis of the affected tissue may occur and produce a distinctly abnormal reduction and alteration in the kidney outline.

Tuberculosis. Discussion of this common type of dystrophic calcification is omitted as most radiologists and urologists are familiar with the clinical and roentgen characteristics of this entity.

Bilharziasis. This is an extremely rare form of renal calcification. Calcium demarcation of the bladder and lower ureter is more commonly observed than of the renal pelvis in cases of bilharziasis (Fig. 2, *A* and *B*) and has been reported by several Egyptian and German urologists.

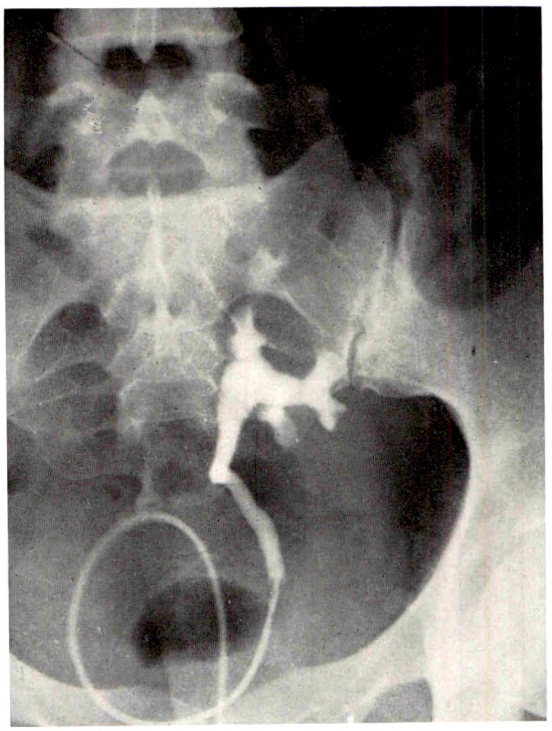


Fig. 1. Normal right kidney and congenital pelvic kidney on the left. No calcification can be detected in the left ectopic kidney, which lies over the sacrum, but diffuse calcium deposits were found at histologic examination of the sections. Diagnosis was chronic pyelonephritis.

Female, aged forty-two, with dull back pain and hypertension, no urinary symptoms, vaginally palpable left kidney, and negative urinalysis. Left nephrectomy was performed.

B. Obstructive Lesions

Hydronephrosis and Pyonephrosis. The hydronephrotic kidney usually consists of a shell of fibrous tissue which exhibits localized or diffuse impregnation with calcium carbonate or phosphate. In the pyonephrotic kidney, a dry semicaseous material containing calcium salts is found in the lining of the abscess cavities and dilated calyces as well as localized deposits of calcium salts in the septa. The extent of the calcification and ossification depends in a large measure upon the duration of the disease and the degree of fibrosis. Some of the apparent stones in the kidney parenchyma prove to be cavities, connected with the renal pelvis through a constricted calyx (localized pyonephrosis or infected hydrocalycosis), containing a mass of calcified blood clots (Fig. 3, *A* and *B*; 4; and 5).

C. Tumors of the Kidney

Cystic Lesions

Solitary Serous Cysts. Calcification is observed only when secondary degenerative changes such as infection, necrosis, or hemorrhage have occurred within the cyst (Fig. 6; and 7, *A* and *B*).

Hemorrhagic Cyst. The occurrence of calcification in the walls of this type of cyst has rarely been reported despite its intimate relation to a serous cyst and trauma. Magoun[17] reported a calcified hemorrhagic cyst in the upper pole of the left

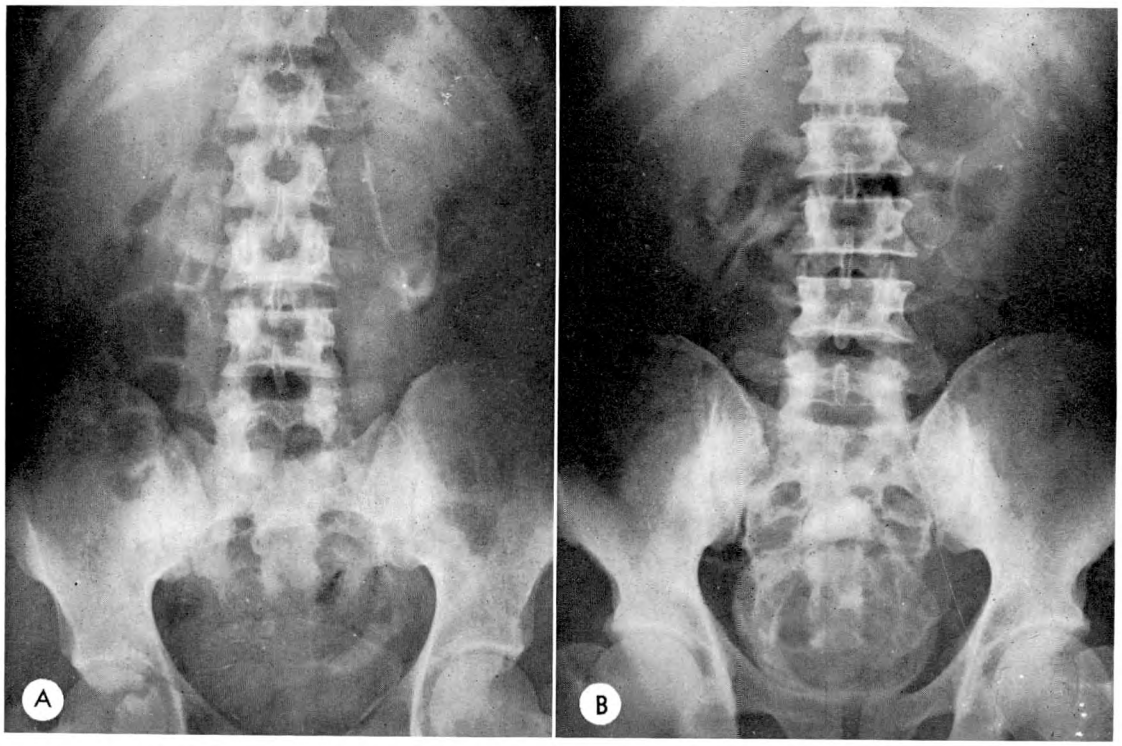


FIG. 2. (*A* and *B*) Plain roentgenograms of 2 different cases of bilharziasis showing the outlines of calcified parasites in the left renal pelvis, left ureter and bladder area. (Courtesy of Dr. Polani.)

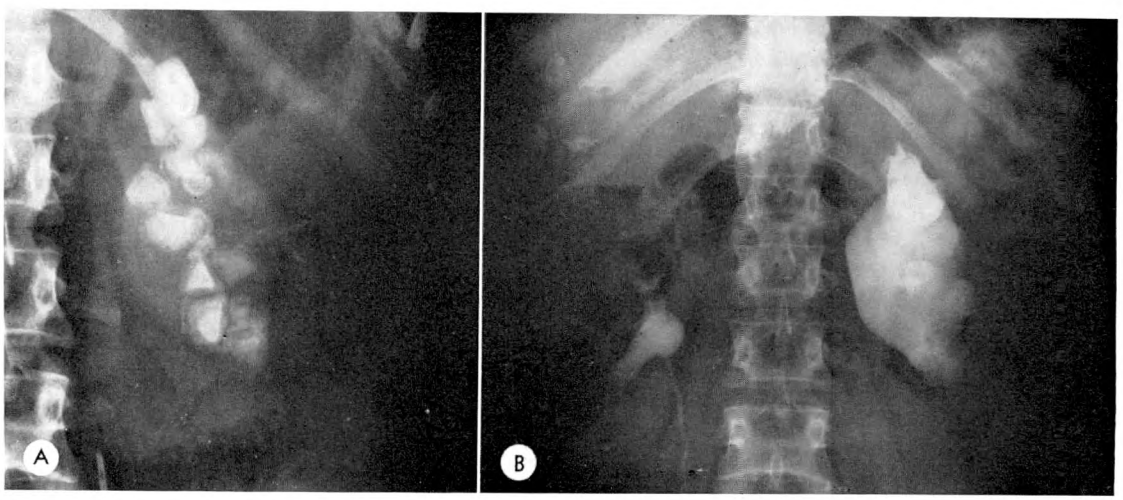


FIG. 3. (*A*) Plain roentgenogram shows multiple stones in the left kidney, which is appreciably enlarged. (*B*) Intravenous pyelogram discloses a huge hydronephrotic sac and evidence of an obstruction at the ureteropelvic junction.

Female, aged thirty-three, with gross hematuria and left lumbar pain. At nephrectomy a large extrarenal pelvis, multiple stones and ureteropelvic obstruction were found. Pathologic report: severe, acute and chronic pyelonephritis, hydronephrosis and multiple renal calculi.

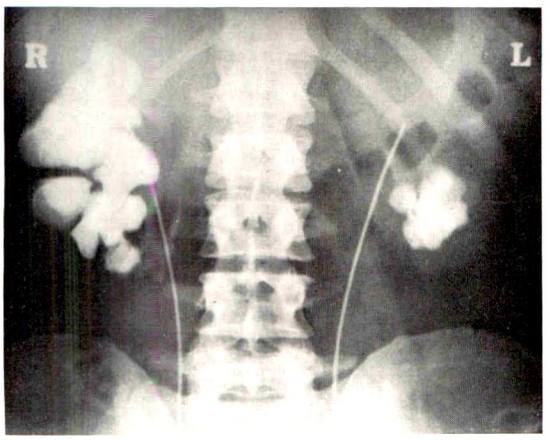


Fig. 4. Multiple stones fill the entire right kidney and the lower part of the left kidney.

Male, aged thirty-one, who had kidney colic four months prior to admission. Right nephrectomy disclosed an extremely dilated pelvis and calyces with the medulla and cortex thinned-out to 0.5 cm. in thickness. Pathologic diagnosis: pyohydronephrosis, chronic pyelonephritis, and nephrolithiasis.

kidney, which also contained a distinctly separate papillary carcinoma in another portion of the upper pole.

Multilocular Cyst. Calcification is a most unusual finding in this rare type of cystic disease.

Peripelvic or Parapelvic Cyst. This rare type appears as a single cyst of varying size, adjacent and adherent to the renal pelvis. Presumably it is due to blockage by a chronic inflammatory process of the lymphatic vessels draining the pelvis, and only 3 cases of calcification of a peripelvic cyst have been reported.

Polycystic Disease. Goldstein and Abeshouse[14] were able to collect from the literature only 3 cases of calcification in a polycystic kidney and they added a personal case (Fig. 8).

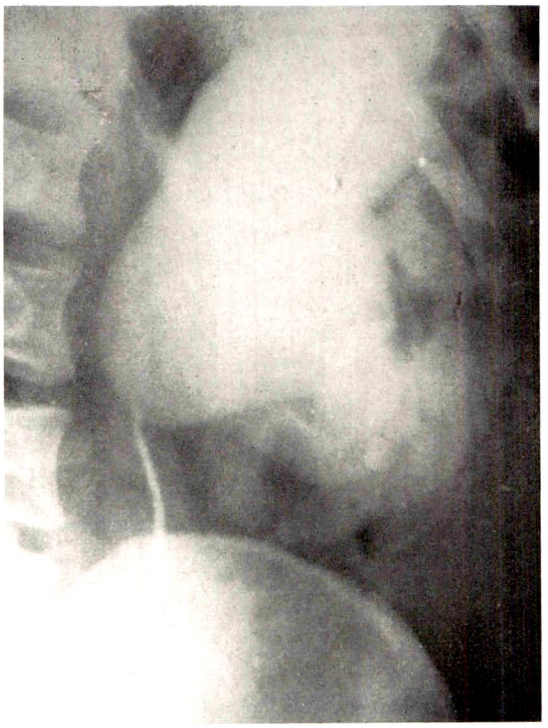


Fig. 5. Plain roentgenogram, with a catheter in the left ureter, showing a large saccular pyonephrosis with scattered flecks of calcium in the dilated calyces due to organization of a clot formation and the deposition of calcium salts.

Male, aged thirty-five, with sharp pain in the left flank and no urinary symptoms. Urinalysis showed occasional pus and red blood cells. Left nephrectomy was performed.

Congenital Unilateral Multicystic Kidney. During the past ten years this unusual clinical and pathologic entity has been recognized more frequently. It is essentially a congenital renal aplasia with cystic

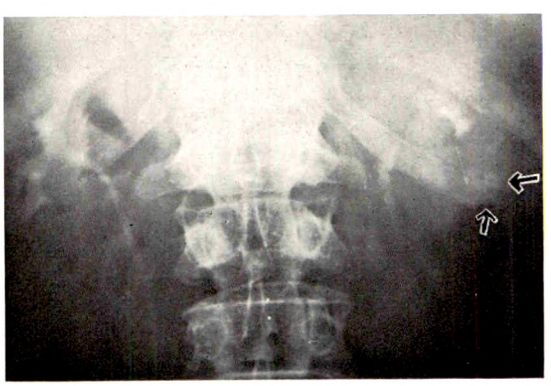


Fig. 6. Intravenous pyelogram discloses a calcified cyst-like mass in the lower pole of the left kidney. The lower calyces are compressed and almost obliterated.

Male, aged fifty-six, with dull left flank pain and a tender palpable mass in that area, a negative urinalysis, and no history of trauma. One large and one small cyst were excised; focal deposits of calcium were found in the wall of the large cyst.

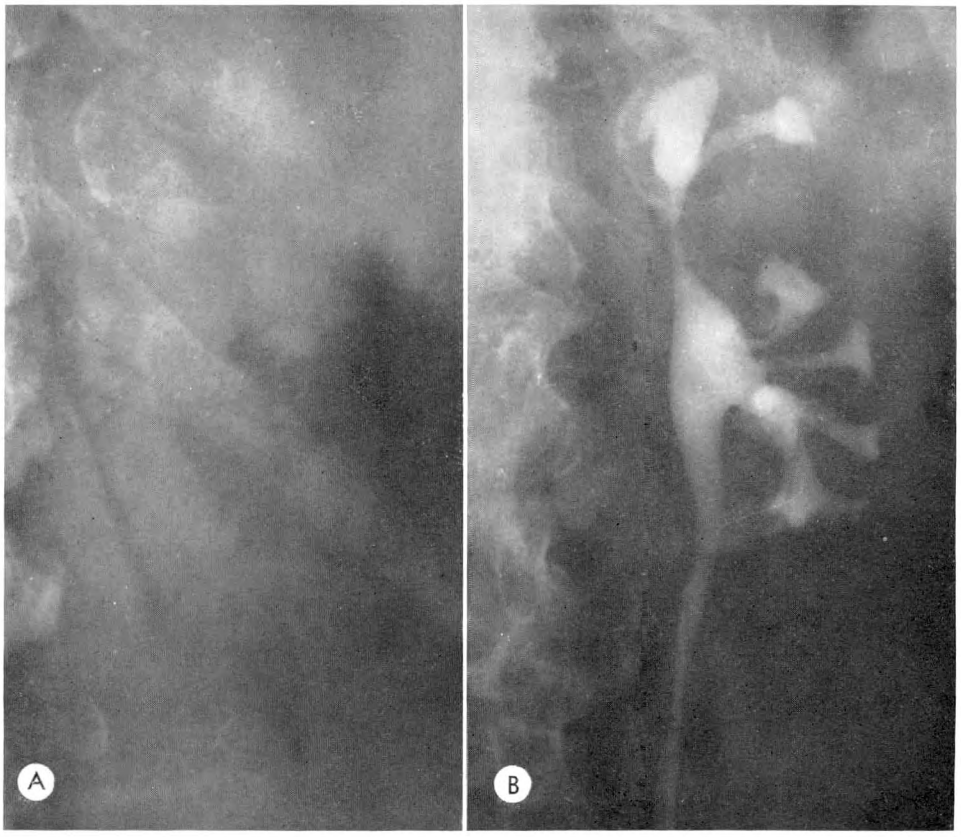


FIG. 7. (*A*) Plain roentgenogram discloses semicircular calcium deposits overlying the upper mesial aspect of the left kidney. (*B*) Intravenous pyelogram shows that this calcification is in the upper portion of the left kidney and that the lateral calyx is somewhat compressed.

The patient came to the hospital for a general check-up and, as part of the work-up, intravenous pyelography was performed with the above findings. At nephrectomy a benign cyst was found. (Courtesy of the Department of Radiology, Johns Hopkins Hospital.)

degeneration resulting from faulty development of the nephrogenic elements of the metanephros. The cysts vary in number and in size. They are scattered throughout the vestigial remnant producing an irregular, lobular, movable, nontender mass which is smaller than the normal kidney. The scattered cysts often have the appearance of a bunch of grapes. Hyalinization, calcification, ossification and cartilage have been found in the walls of the cysts or in the fibrous septa between the cysts. These secondary changes apparently have no specific predilection for either age or sex and are most frequently observed in the fourth, fifth, and sixth decades. Friedman and Abeshouse[13] collected 78 cases, including 5 personal cases, and found calcification in 25 and ossification in 4 (Fig. 9, *A* and *B*).

Echinococcus Cyst. This is the most common cystic disease of the kidney to exhibit calcification in the cyst walls. The degenerative and retrogressive changes in the cyst walls predispose to the deposition of calcium salts In recent literature, several reports on this subject appeared by Berger and Cowan,[7] Teplick *et al.*[19] and others.

Sponge Kidney. This condition is essentially a congenital malformation of the renal pyramids manifested by cystic dilatations of the collecting tubules. The calcification observed in reality represents calculi within the cystic cavities and should be classified with nephrocalcinosis rather than as a dystrophic manifestation (Fig. 10, *A*, *B* and *C;* and 11).

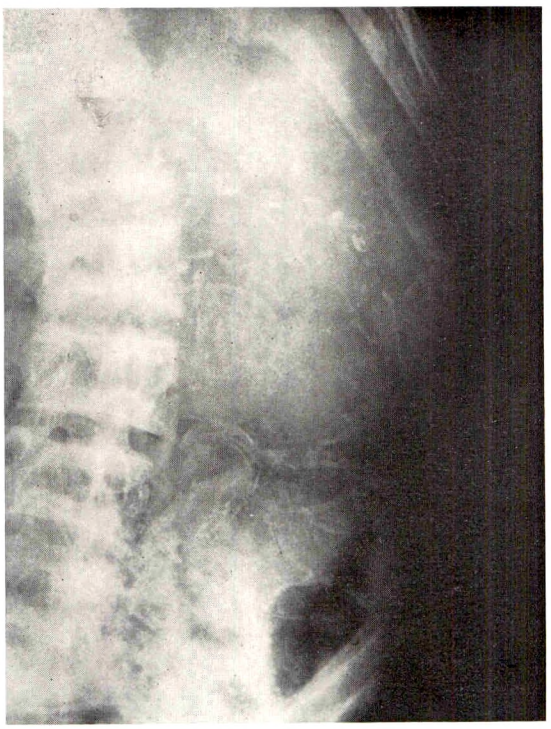


Fig. 8. Roentgenogram in an advanced case of renal rickets discloses marked bony changes and calcification of vessels. There are diffuse calcium deposits in the kidney area with large calcified cysts in the left kidney.

Female, aged twenty, in whom a diagnosis of polycystic disease had been made at the age of sixteen. Other clinical findings were myocardial disease, electrolyte imbalance, acidosis and uremia. The patient died and autopsy was refused.

⟿

BENIGN TUMORS

Adenomata may be classified histologically as (a) tubular, (b) papillary and (c) alveolar. These tumors frequently exhibit secondary changes resulting from degeneration, necrosis, and hemorrhage which may be manifested as cystic degeneration. The cystic changes vary considerably and may impart a honeycomb or polycystic appearance (Fig. 12; 13, *A* and *B;* and 14, *A* and *B*). In an occasional case the cyst may attain a large size and possess thick walls; if this occurs, the calcification may be visible on the roentgenogram. Allen,[3] Watts,[20] and Bauer *et al.*[6] reported such cases which were treated by surgery, and the present authors had a similar case. The authors also have had a case of a calcified papilloma of the renal pelvis which was mistaken for a calculus in the pelvis and treated by nephrectomy (Fig. 15, *A* and *B*).

MALIGNANT TUMORS

Pathogenesis of Calcification and Ossification. In malignant tumors of the renal parenchyma this is intimately related to

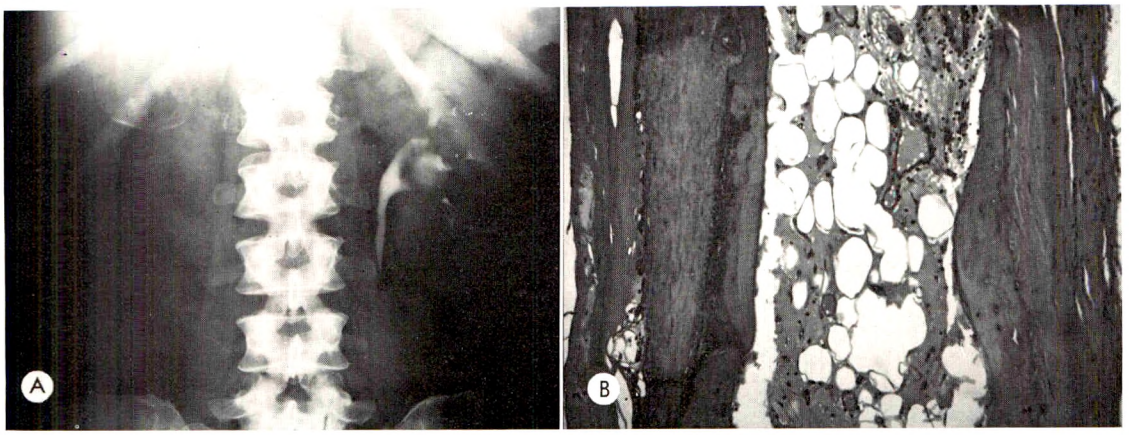


Fig. 9. (*A*) Intravenous pyelogram shows a well-functioning left kidney and apparently a nonfunctioning right kidney. Three cystic structures lined by calcium deposits are seen in the right kidney. The largest is in the upper pole; the two smaller are below. (*B*) A section through the cyst wall shows a broad band of osseous tissue covered on both the inner and outer aspects by compact, somewhat acellular connective tissue.

Female, aged forty, with dull pain in the right upper quadrant and pyuria. Right nephrectomy was done for congenital aplastic kidney with calcified cystic degeneration and ossification.

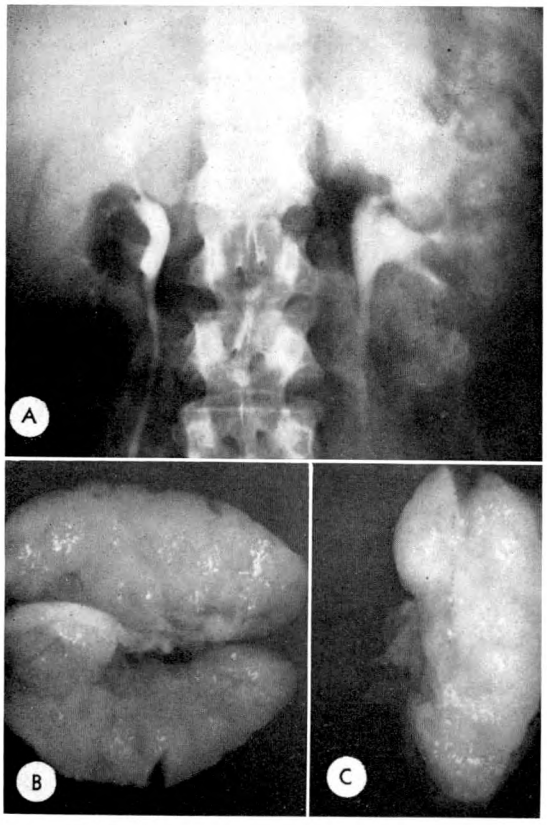


FIG. 10. (*A*) Bilateral pyelogram shows numerous small calcium deposits in the pyramids of both kidneys. The pelves and calyces appear to be normal. (*B* and *C*) Photographs of the left kidney operative specimen show the same findings.

Female, aged forty-five, with dull pain in the left kidney area and pyuria. The urine contained numerous pus cells and *B. coli*. There was a marked reduction in function of the left kidney. At left nephrectomy the kidney showed diffuse medullary cysts with numerous calculi characteristic of sponge kidney and chronic pyelonephritis.

the development of degenerative and retrogressive changes within the tumor. These changes are manifested as necrosis, hemorrhage, and cystic degeneration and they are present to some degree in every so-called hypernephroma and to a lesser degree in the so-called adenocarcinoma of the clear cell or granular cell type. The extent of these changes varies with the size and rate of growth of the tumor. In the pseudoalveolar and papillary types of hypernephroma, a large pseudocyst may develop as a result of necrosis and cystic degeneration. In the solid type of hypernephroma, which is composed of a densely packed nest of cells, areas of fresh hemorrhage may be present as well as cystic areas lined by organized blood clots or blood pigment. The necrobiotic process resulting from the pressure and vascular changes induced by these degenerative lesions may be confined to one or more areas and occasionally involves the capsule of the tumor. As time passes, secondary degenerative changes, such as hyalinization and calcification, and metaplastic changes, such as ossification and cartilage formation, may develop.

Roentgen Appearance. Roentgenographi-

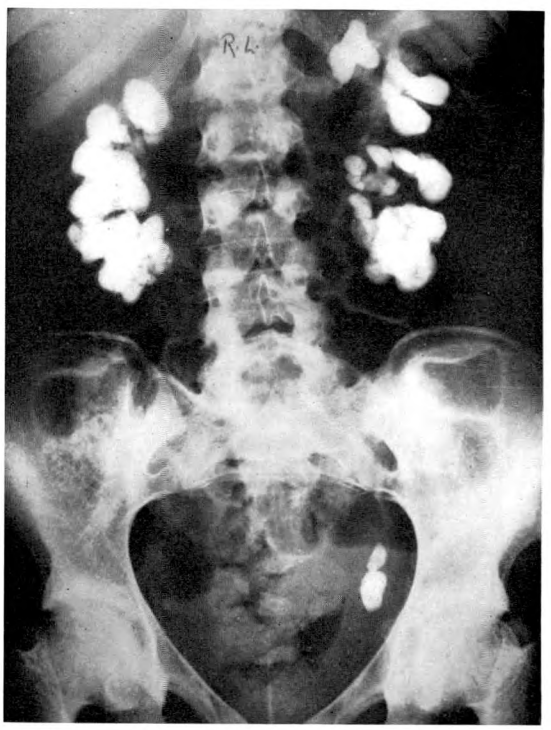


FIG. 11. Multiple small bilateral renal calculi in a case of renal rickets. The calcium shadows are definitely located in the pyramids immediately adjacent to the terminal calyces and are composed of clusters of small round discrete calculi. The primary renal disease is regarded as being congenital cystic dilatation of the collecting tubules or "sponge kidney." The calculi are a secondary complication. Note a large stone in the lower left ureter.

Female, aged twenty-one, who underwent three operations to correct bone deformities in the past eleven years. Renal rickets was diagnosed three years ago and two small calculi were passed in the last two years. The patient developed acidosis and uremia and died. Autopsy was refused.

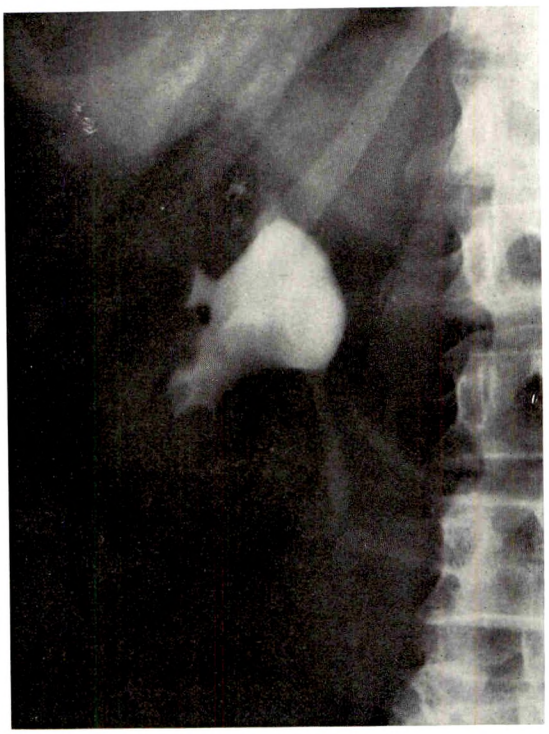


Fig. 12. Right retrograde roentgenogram discloses an irregular filling defect in the superior minor calyces with several calcific densities.

Female, aged sixty-six, with a history of pain in the right costophrenic angle and occasional hematuria of six weeks' duration. Right nephrectomy was done. Pathologic diagnosis: well-differentiated, noninvasive adenoma. (Courtesy of the Department of Radiology, Johns Hopkins Hospital.)

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cally, the calcium deposits exhibit great variation in size, configuration, and position. They may be classified, according to the roentgen appearance, into five groups: (1) the most common is a single calcified area which appears as one or more irregular streaks or spots (Fig. 16); (2) next in frequency, the calcified area may appear as a network of irregular strands (Fig. 17, *A* and *B*); (3) less frequently, the calcified area may assume the shape of an irregular plaque (Fig. 18, *A* and *B*) or whorl (Fig. 19, *A* and *B*); (4) in an occasional case, the calcification appears chiefly to involve the capsule of the neoplasm and may or may not be accompanied by one or more areas of calcification in the body of the tumor; and (5) in rare instances, the calcification appears as an incrustation on a papillary-shaped mass of tissue projecting into the pelvis.

Prognostic Significance. There is a decided difference of opinion regarding the prognostic significance of calcification within the malignant neoplasm. Braasch and Griffin[8] studied 7 cases of calcified hypernephroma treated by nephrectomy and found that all but 1 lived more than

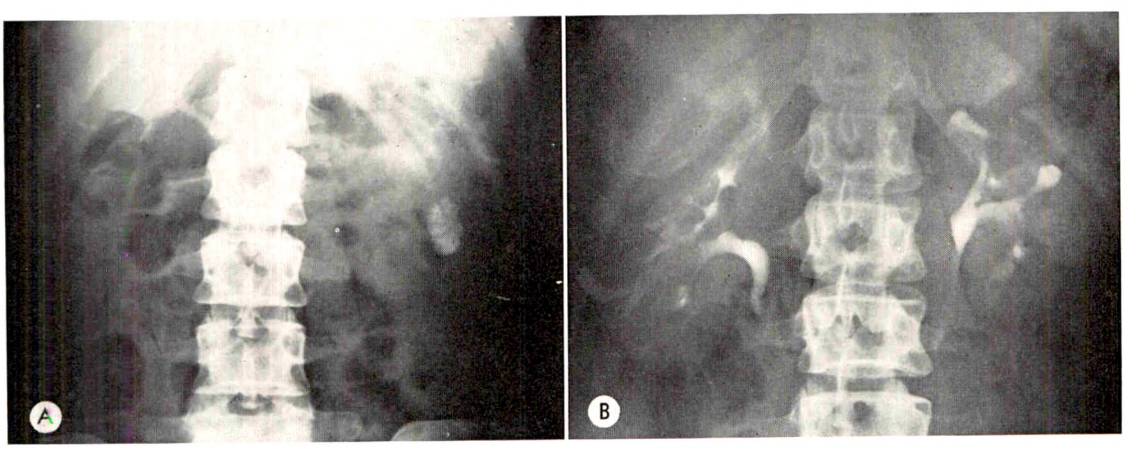


Fig. 13. (*A*) Scout roentgenogram and (*B*) intravenous pyelogram show an amorphous accumulation of calcifications in the left lower kidney.

Female, aged thirty-two, with a history of left flank pain of twelve years' duration. Initially, she had been treated for urinary infection and left kidney stones but she continued to have intermittent left flank pains and the size of the calcified mass increased on roentgenographic studies. Left nephrectomy revealed a mass containing calcified material. (Courtesy of Dr. Frederick F. Ruzicka.) Pathologic report: epidermoid cyst of the kidney containing gray-brown calcifications and yellow-white necrotic remnants of renal tissue. (Courtesy of Dr. John Alfred Wagner.)

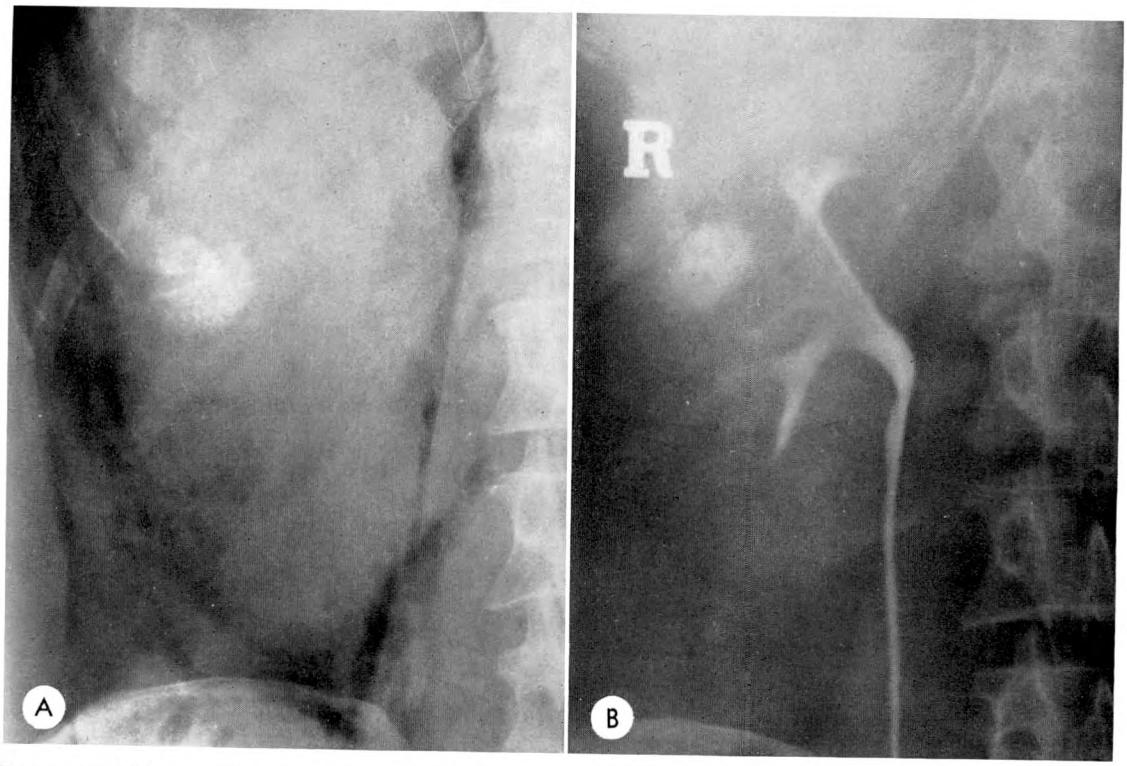


FIG. 14. (*A*) Perirenal insufflation roentgenogram and (*B*) retrograde pyelogram show a large right kidney with a calcified round lesion in the upper and lateral aspect of the kidney substance.

Male, aged sixty, with a history of a right kidney stone removed fifteen years ago and hematuria one week prior to admission. Right nephrectomy revealed a calyceal diverticulum containing calculi. This condition cannot be differentiated roentgenologically from benign tumors with calcifications.

three years and 4 were alive after five years. They concluded that calcification was a favorable prognostic sign. Subsequent studies by other investigators failed to support this contention. Cahill and Melicow[9] reviewed 118 renal neoplasms and found evidence of calcification on the plain roentgenogram in 14 of 82 cases of parenchymal (hypernephroid) tumors, but only 1 of 12 nephrectomized patients survived two years without demonstrable metastases. Abeshouse and Weinberg,[2] in a review of 63 malignant neoplasms, observed calcification in only 8 of 56 cases of parenchymal tumors. Three nephrectomized patients survived one, two and three years, respectively, and a fourth patient with bilateral hypernephromata lived eleven years after the removal of a noncalcified tumor bearing kidney. Two patients were not operated upon because of demonstrable pulmonary metastases and died within one year.

Austen[4] reviewed 131 malignant neoplasms and excluded 25 cases (3 showed calcification) not verified by pathologic examination. In the remaining 106 cases, calcification was present in only 1 of 8 benign tumors and in 16 of 98 malignant tumors. The five year survival rate was 15.3 to 18.7 per cent as compared with 28 to 33 per cent in the group without metastases. The majority of patients with calcification in malignant tumors died within four to forty-eight months following operation. In recent years a number of reports have appeared on nephrogenous polycythemia in the presence of renal neoplasms[10,12,15] (Fig. 19, *A* and *B*).

Osteosarcoma (primary and secondary). Roentgenologically, these tumors appear as large irregular calcified masses in and

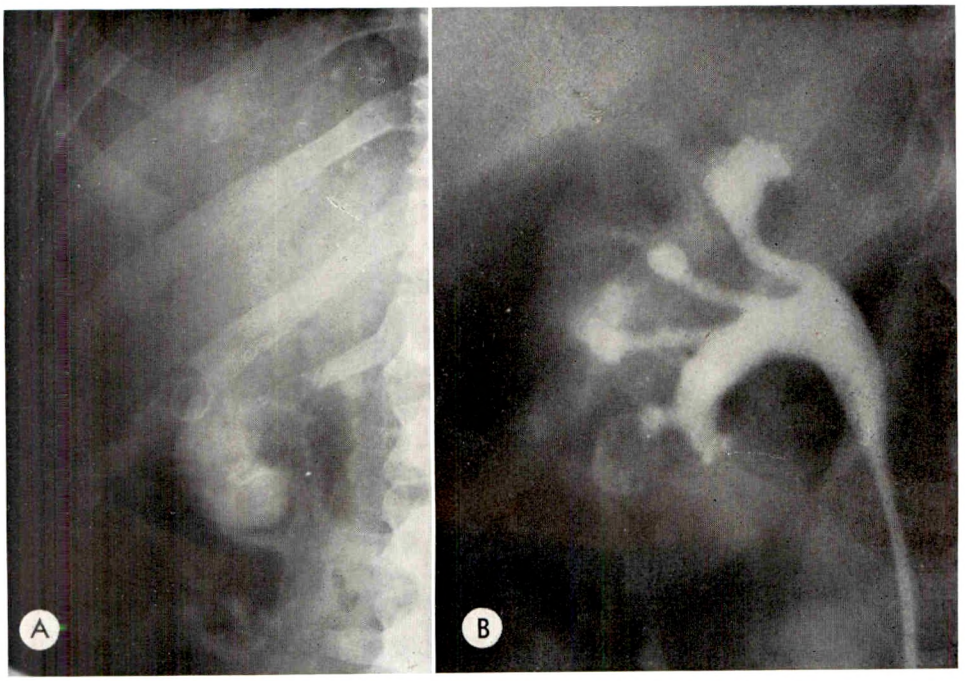


FIG. 15. (*A*) In the right upper quadrant a coin-shaped mottled calcium deposit is projected through the abdomen and outside the gallbladder area. (It could imitate a gallstone.) (*B*) Stereoscopic retrograde pyelogram reveals that this collection of calcium is within the lower pole of the right kidney, but not attached to or involved in the pelviocalyceal system.

Male, aged fifty-nine, with a diagnosis of prostatism. The above finding was discovered during a routine work-up. Partial right nephrectomy was done. Pathologic diagnosis: calcified adenoma of the papillary type.

around the affected kidney. Histologically, they show a typical sarcomatous pattern interrupted with osteoblastic elements producing bone and occasionally a small amount of cartilage. The primary tumors originating in or about the kidney are presumably due to a metaplasia of fibroblasts.

Osteoma (*pararenal teratoma, dermoid cyst*). The tumor is usually pararenal in location, is believed to be derived from epidermal rests of wolffian origin, and is

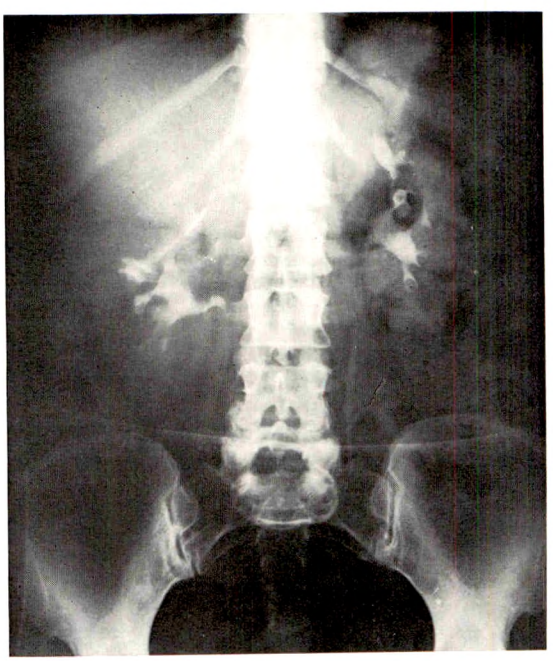


FIG. 16. Intravenous pyelogram discloses a mass in the upper part of the right kidney compressing the upper calyceal system. Two small calcifications are seen within this mass.

Female, aged fifty-four, with a history of right lower quadrant pain and gross hematuria of one week's duration. At surgery a multilobular hypernephroma replacing the upper pole of the right kidney was found. (Courtesy of the Department of Radiology, Johns Hopkins Hospital.)

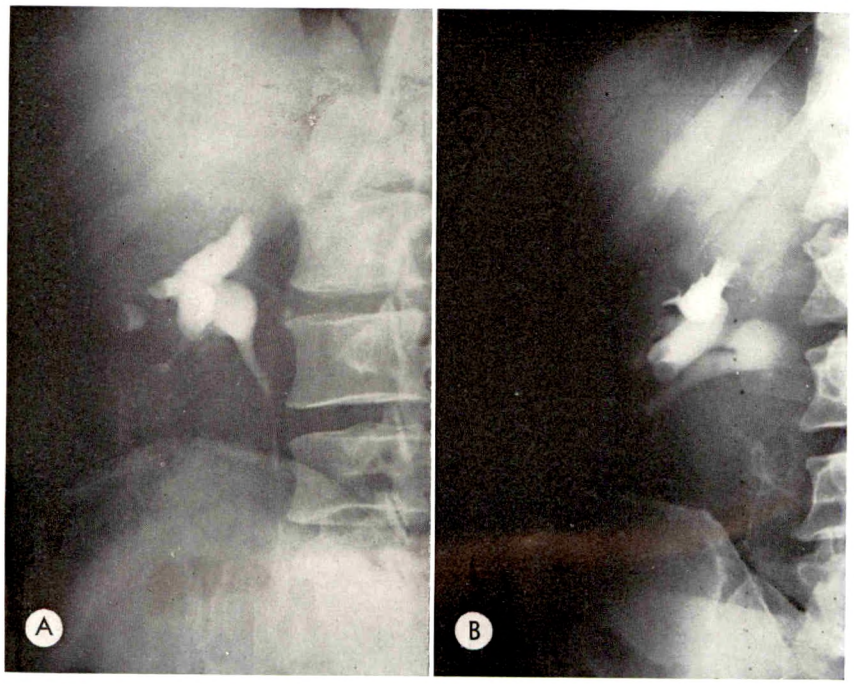


FIG. 17. (*A* and *B*) Intravenous pyelograms show a large mass occupying the lower half of the right kidney. The mass contains a network of calcifications and compresses and displaces the lower calyces and pelvis.

Male, aged fifty-eight, with a palpable mass. Examination of the specimen disclosed a hypernephroma containing calcifications.

attached to or invades the kidney to produce a bulging mass under the capsule of the kidney (Fig. 13, *A* and *B*). The outstanding histologic feature is the presence of bone and calcification within the tumor. The tumor is regarded as benign.

It is extremely difficult at times to distinguish between an osteoma or dermoid cyst of the kidney and a pararenal or retroperitoneal osteoma or teratoma. The major-

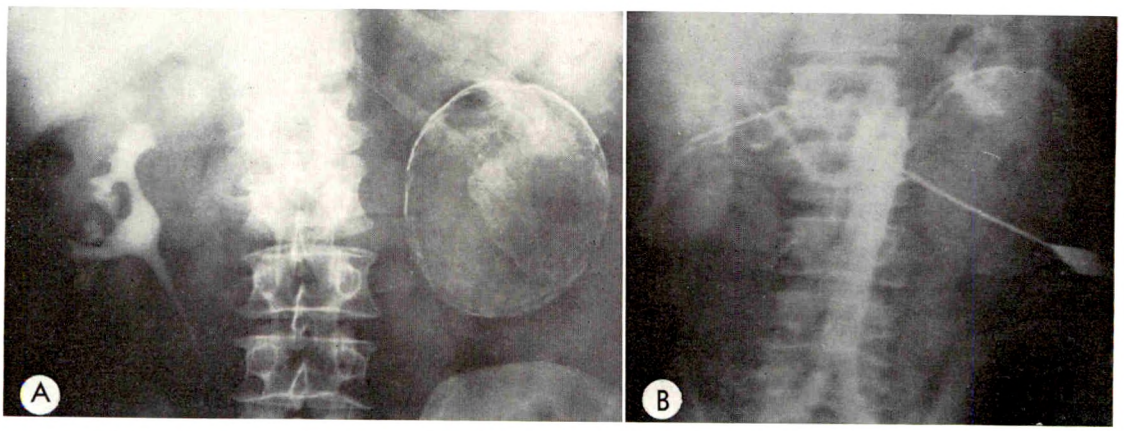


FIG. 18. (*A*) Excretory urogram showing a large calcified cyst situated within a large hypernephroma of the upper pole of the left kidney. No contrast medium was excreted within the left kidney. The right kidney is normal. (*B*) Translumbar aortogram showing a normal blood supply to the right kidney. On the left side the blood supply was not demonstrated. The calcified cyst in the center of the left hypernephroma is clearly defined.

Male, aged fifty-two, who presented with dull pain on the left side and poor general condition. Autopsy showed a large calcified cyst within a hypernephroma of the left kidney.

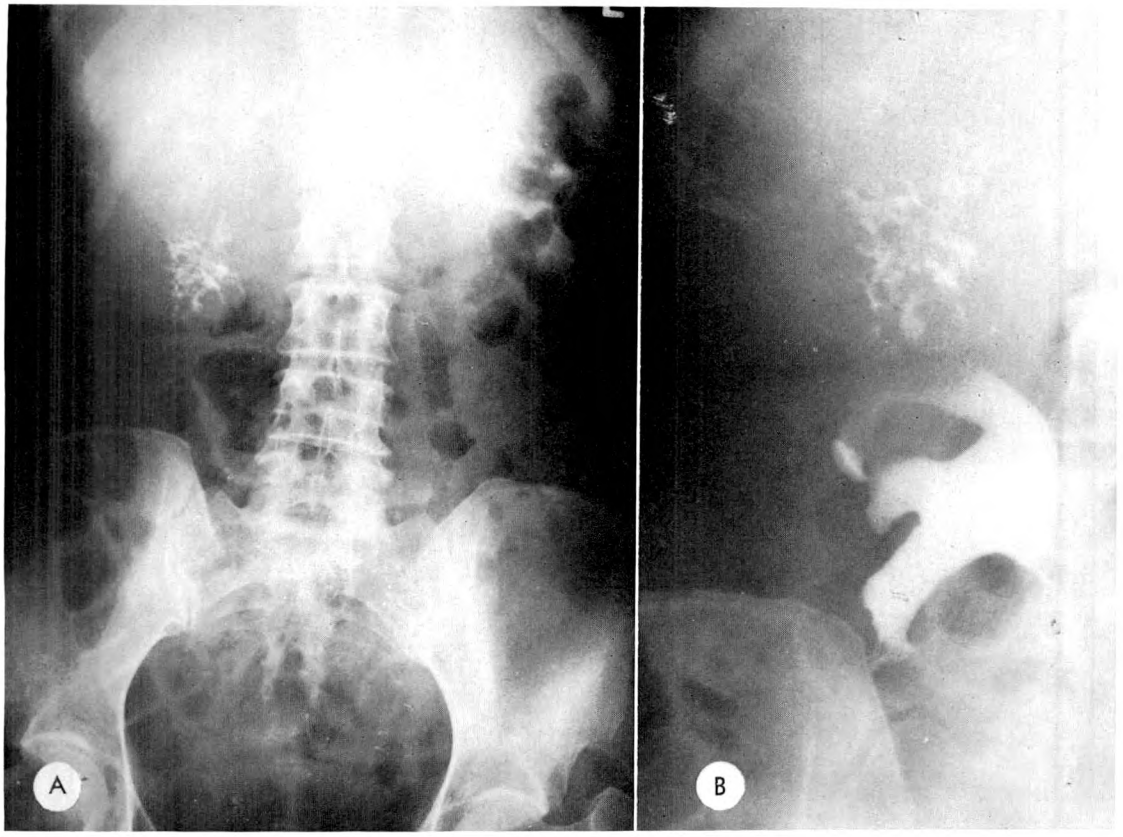


Fig. 19. (*A*) Scout roentgenogram of the abdomen and (*B*) retrograde pyelogram show a large mass in the upper pole of the right kidney splaying and compressing the upper calyces. The mass contains a network of irregular calcifications giving a strand-like and whorl-like appearance.

Female, aged forty-five, was referred because of pink eye to an eye specialist who, in turn, asked for a medical check-up which disclosed the presence of apparently secondary polycythemia. Further examination revealed the right kidney mass, which was removed. Pathologic diagnosis: hypernephroma.

ity of cases of pararenal osteoma are found in children or young adults. In some cases an antecedent history of trauma is obtained. Not infrequently, the areas of ossification and calcification are mistaken for renal calculi when these shadows are over the kidney outline. The extent of involvement of the kidney appears to be the chief point of differentiation. The majority of pararenal osteomas or teratomas are situated in the retroperitoneal area and, although they may be independent of the kidney, in most cases they are attached to the kidney at one of the poles.[5,11]

Miscellaneous Malignant Tumors. The so-called "mixed" tumor or teratoma of the kidney is essentially a Wilms' tumor; it occasionally contains teeth, bone and calcification in addition to tissue of endodermal, mesodermal or ectodermal origin.

Cholesteatoma. There are two types of cholesteatoma: (1) primary, which is a true neoplasm; and (2) secondary, which is a false or pseudotumor composed of keratin mass found in any body cavity as a result of chronic inflammation or irritation. The pathologic process is essentially that of a squamous metaplasia and occasionally is associated with leukoplakia. In the kidney, cholesteatoma simulates, and frequently is mistaken for, a squamous cell carcinoma. Occasionally, the cholesteatomatous mass contains deposits of calcium salts, but the important roentgenographic findings are filling defects in the pelvis or calyces.

Adrenal Diseases. It is generally agreed

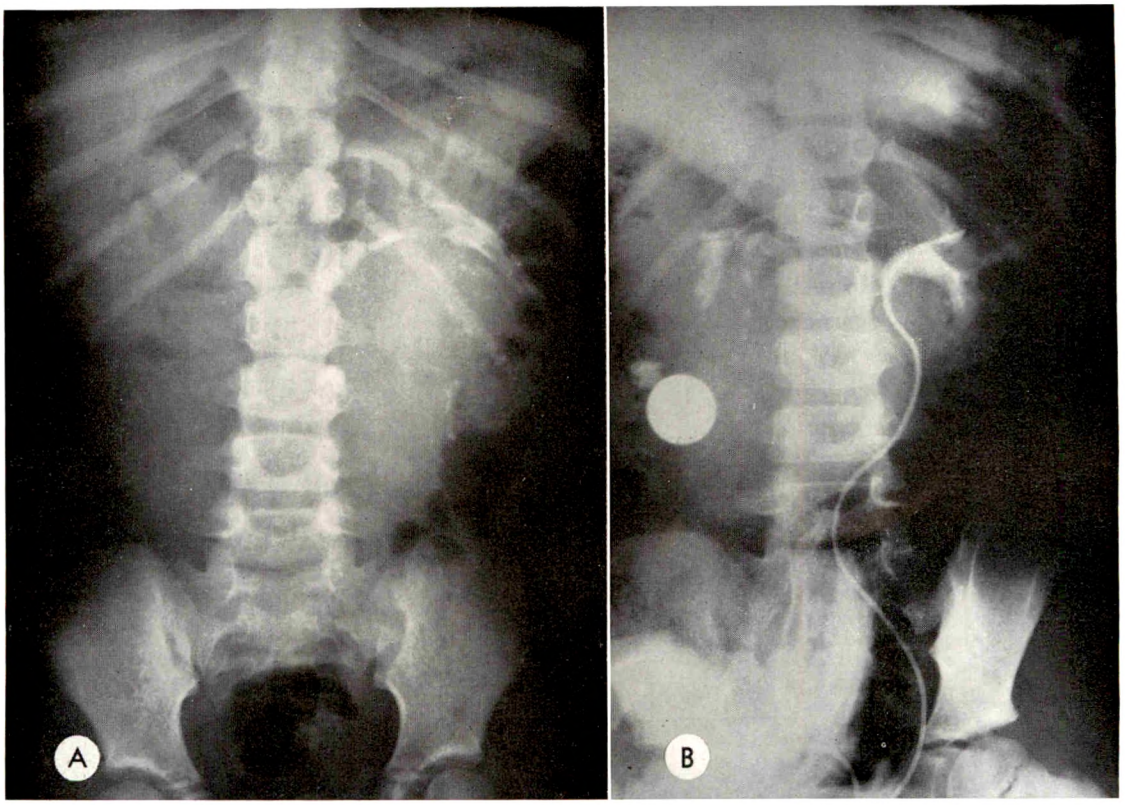


FIG. 20. (*A*) Abdominal roentgenogram shows a mass, projected through the upper abdomen, containing numerous calcifications, mainly on the left side. (*B*) Left retrograde pyelogram in the oblique view shows the lateral and anterior displacement of the left ureter. A coin is placed over the main mass.

Female, aged six, had a palpable asymptomatic mass. A ganglioneuroma was removed from the tetroperitoneal area and the kidney was preserved. Several discrete areas of calcification were present within the tumor.

that the most common type of calcification of the adrenal gland is due to tuberculosis associated with Addison's disease. Calcification and ossification are rarely observed in adrenal tumors such as carcinoma, pheochromocytoma and basophil adenoma, but neuroblastoma, which may originate in the adrenal gland and invade the kidney, frequently shows flocculent areas of calcification within the tumor at the site of necrosis and hemorrhage. Such calcification is rare in patients with Wilms' tumors. One of the authors (B.S.A) recently collected 152 cases of adrenal cysts of all types, which included 3 personal cases, and reported calcification in 10 cases. Calcification is more common in the pseudocysts of the hemorrhagic type than in the lymphatic or serous cysts. The authors observed calcification in the wall of a lymphatic and a pseudocystic hemorrhagic adrenal cyst (Fig. 20, *A* and *B;* and 21, *A* and *B*).

Ureteral Diseases. Calcification, ossification and cartilage formation very rarely accompany ureteral diseases.

D. TRAUMATIC INJURIES

Calcification and ossification may develop following injuries to the kidneys, and this probably occurs more often than present reports indicate. These conditions are of two types—intrarenal and extrarenal.

INTRARENAL

Some of the larger collections of blood within the parenchyma in the subcapsular space or in the perirenal tissue are not always entirely absorbed, and, subsequently, calcium salts may be precipitated from the organized blood clot and be deposited

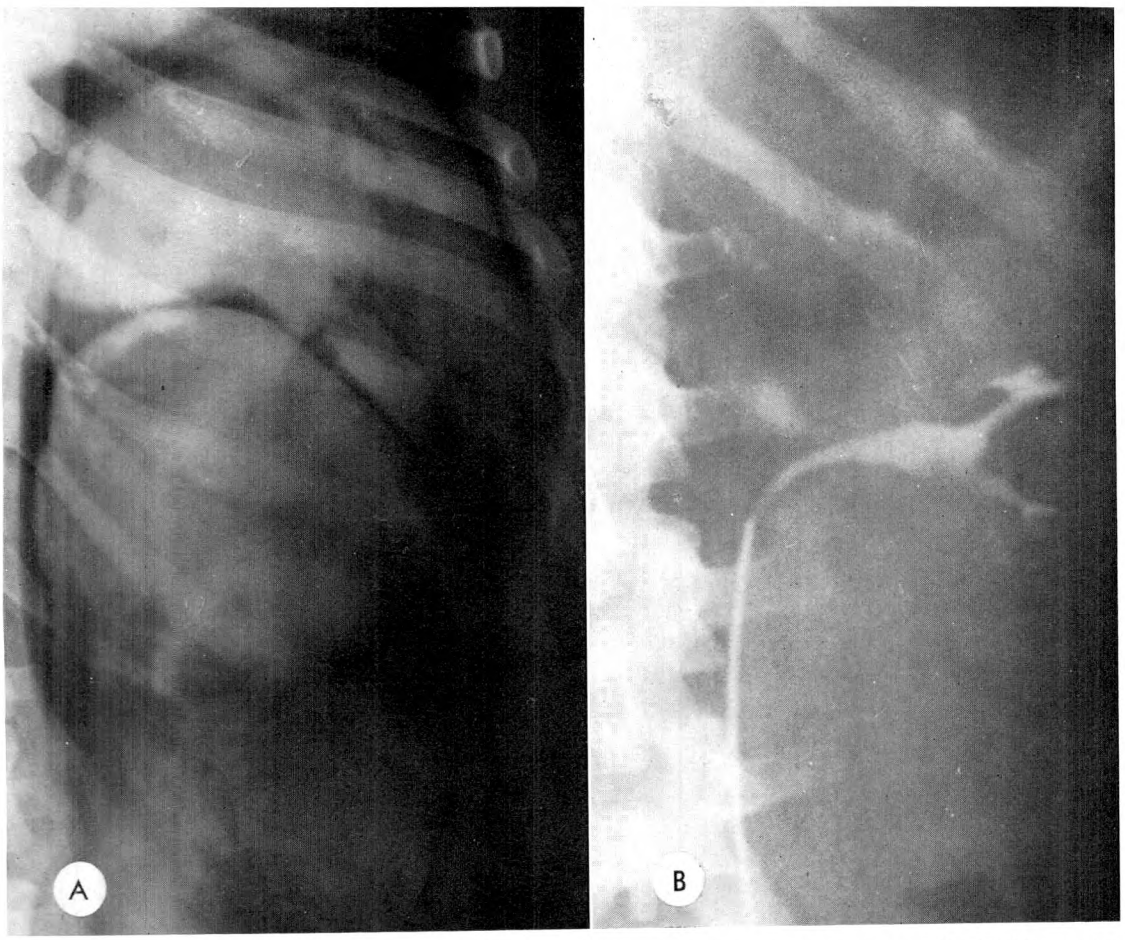


Fig. 21. (*A*) Perineal air injection delineates the cyst. Note calcified areas in the cyst wall. (*B*) Retrograde pyelogram shows downward displacement of the left kidney by a calcified adrenal cyst. Note the calcified areas in the cyst wall.

Female, aged twenty, complained of left abdominal pain. The physical examination and urinalysis were negative. A lymphangiomatous cyst of the left adrenal gland which showed several areas of calcification in the wall of the cyst was removed surgically.

within the damaged parenchyma or in the walls of a subcapsular or perirenal cyst.

EXTRARENAL

Perirenal Hematoma. The extent of the perirenal hematoma and the interval of time elapsing before establishing the diagnosis are the chief factors determining the amount of perirenal calcification. In those cases in which the perirenal hematoma is not absorbed but undergoes coagulation, the calcification, and less frequently the ossification, may appear as an irregular area of plaque formation adjacent or intimately adherent to the kidney. In an occasional case of traumatic rupture of the pelvis, the extravasation of urine and blood in the peripelvic tissues may be followed by encapsulation and calcification of the walls of the cyst-like space which has been formed (Fig. 22, *A* and *B*).

Spontaneous (nontraumatic) perirenal hemorrhage may occur in renal or blood vessel diseases and blood dyscrasias. It was fully described by Wunderlich[21] in 1856. In 1933, Polkey and Vynalek[18] collected 178 cases of spontaneous renal and perirenal hematoma and noted the following sequelae: renal blood cysts in 23 cases (11 per cent), perirenal blood cysts in 6 cases (3 per cent), and perirenal hematchydronephrosis in 8 cases (4 per cent).

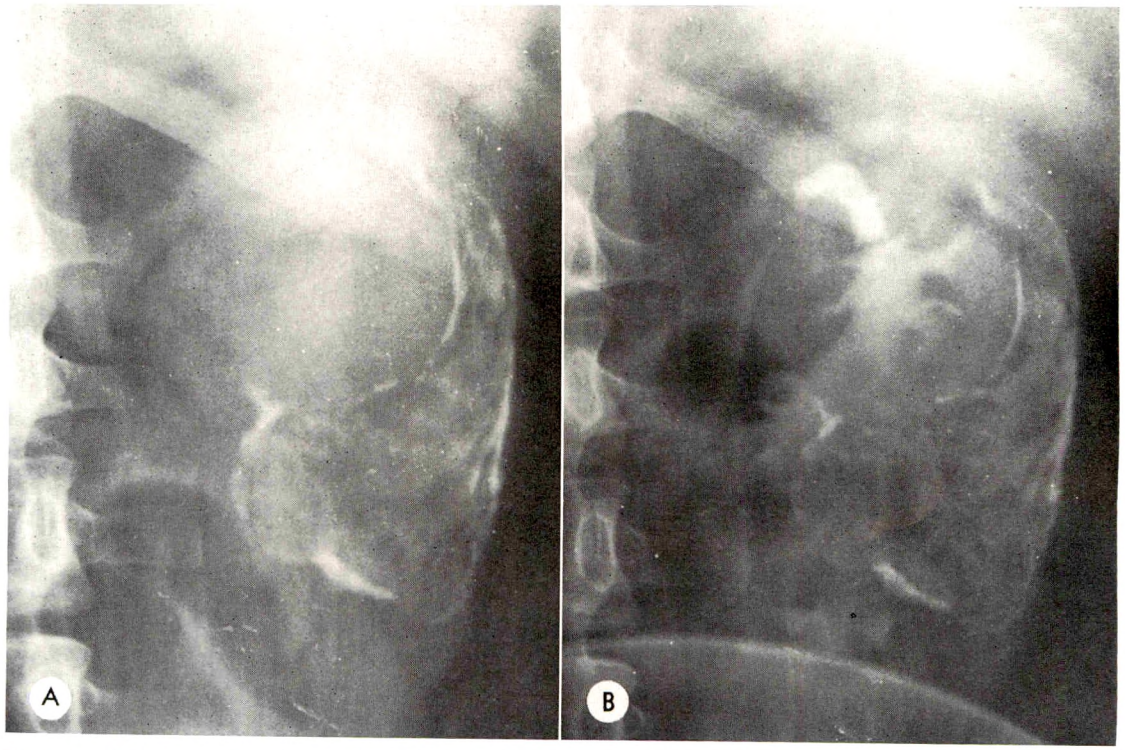


FIG. 22. (*A*) Plain roentgenogram shows large mottled calcium deposits collected around the lower and lateral aspect of the left kidney. (*B*) Intravenous pyelogram shows the same calcified mass around the lower and lateral pole of a left double kidney.

Male, aged forty-four, complained of sharp left renal pain. There was a palpable mass in the left loin. The patient had sustained an injury to the left kidney twenty-two years previously in a football game. The left kidney was removed. Pathologic diagnosis: calcified cystic hematoma attached to the lower pole of the left kidney.

Perirenal Cyst. Large perirenal hematomas may become encysted and show extensive calcified changes in the walls of the cyst (Fig. 23).

Pseudohydronephrotic Cyst (subcapsular hematoma). Occasionally the laceration due to injury may extend through the parenchyma into a calyx leaving the capsule intact. The blood and urine escape through the cortex under the capsule, and a cystic dilatation designated as a perirenal



FIG. 23. Retrograde pyelogram shows lateral displacement of the left kidney by a large perirenal hemorrhagic cyst containing organized blood clots and focal deposits of calcium in the walls of the cyst, especially visible in the lower lateral aspect.

Male, aged forty-five, was admitted in a stuporous condition with a history of persistent hematuria for the past month. A mass was present in the left loin. Autopsy revealed a large perirenal hemorrhagic cyst with several areas of calcification in the cyst wall.

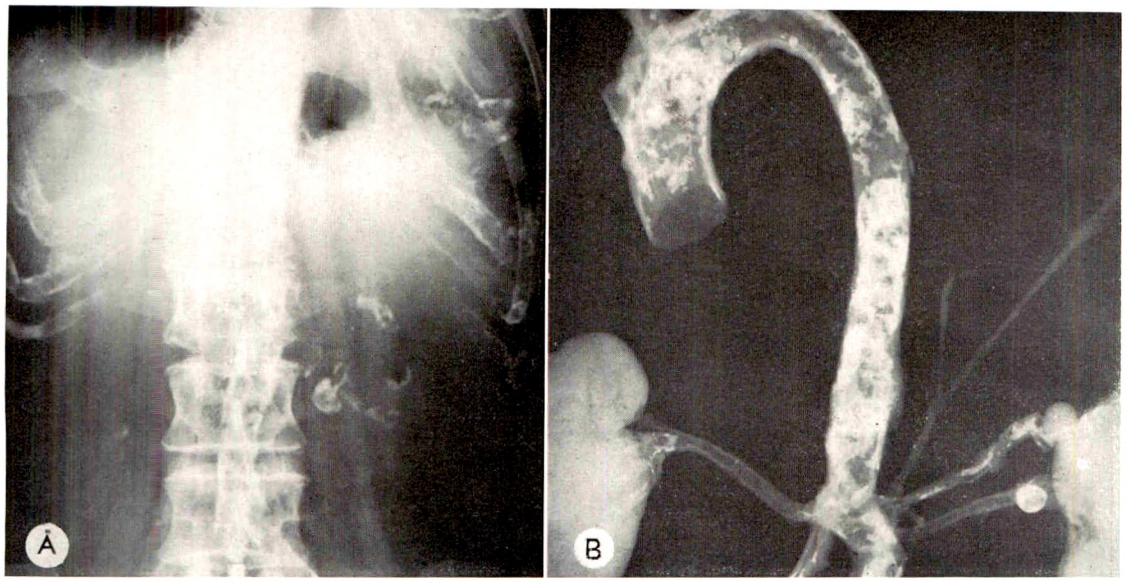


Fig. 24. (*A*) Flat roentgenogram of the abdomen demonstrates the characteristic pattern of a renal aneurysm on the left side. (*B*) Autopsy specimen shows the location of the aneurysm.

or pararenal pseudohydronephrotic or perinephric cyst takes place.

E. VASCULAR DISEASE

Arterial Calcification. This pathologic change is observed in two types of arteriosclerosis: (1) atherosclerosis, wherein the calcification is limited to the intima: and (2) Mönckeberg's disease, wherein the calcification is chiefly in the media. The calcifying process is not exclusively limited to the main renal artery, but may involve the large and small intrarenal branches. Calcification of the large and small renal arteries has also been observed in other conditions, such as neoplasm, renal lithiasis, chronic nephritis, etc. It may also involve the renal vein in rare instances.

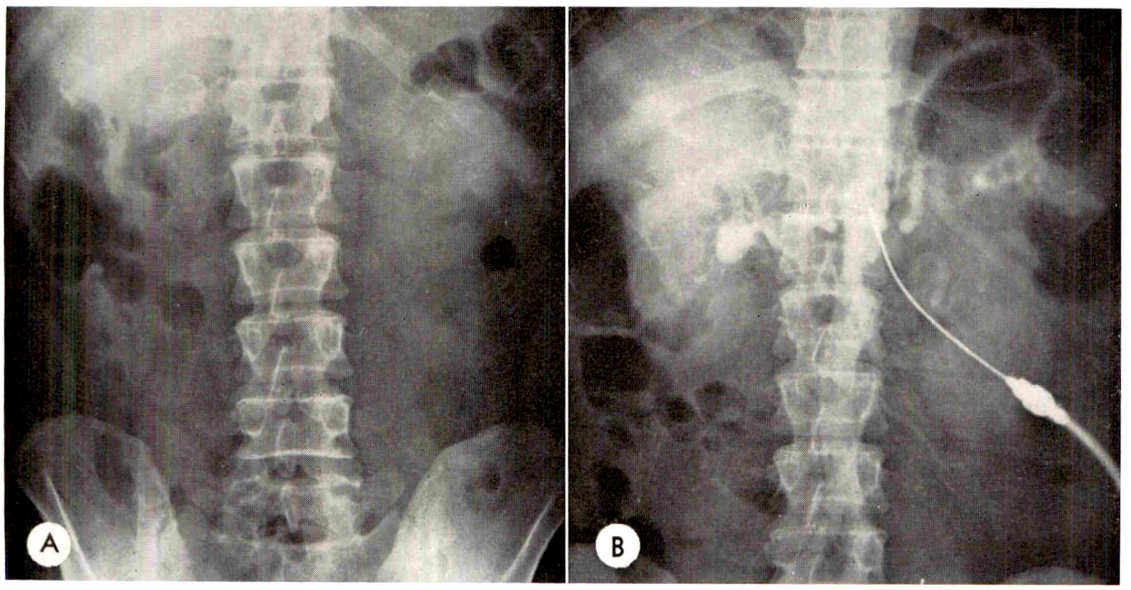


Fig. 25. (*A*) Flat roentgenogram of the abdomen discloses a ring-like calcification in the right upper abdomen. (*B*) Aortogram confirms the presence of a renal aneurysm.

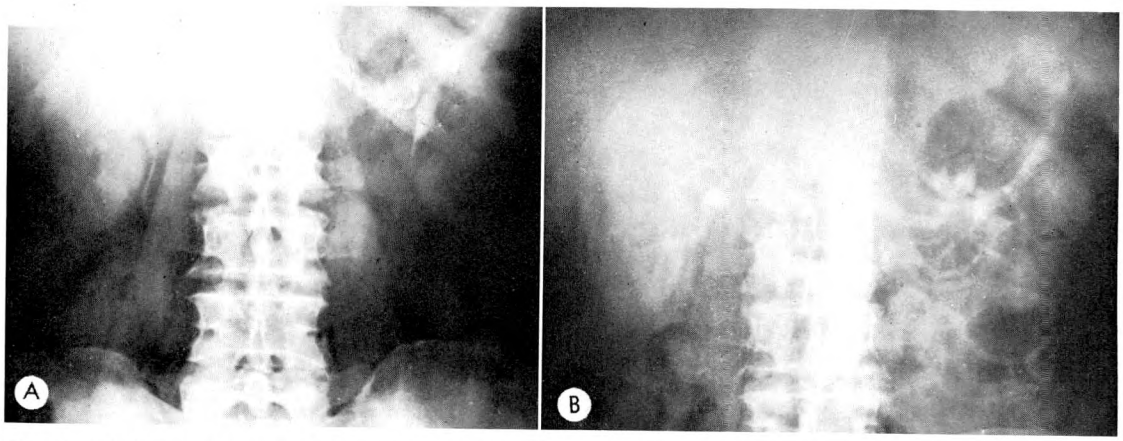


FIG. 26. (*A*) Intravenous pyelogram demonstrates an aneurysm on the left side. (*B*) Aortogram visualizes a small renal aneurysm on the right side in addition to the larger, markedly calcified aneurysm on the left side which was seen on routine excretory urograms.

Aneurysm of the Renal Artery. Calcification is more frequently observed in the true than in the false type of aneurysm of the renal artery. One of the most important diagnostic aids is the presence of a calcified "signet ring" or "wreath-like" shadow along the medial border of the upper pole of the affected kidney (Fig. 24, *A* and *B*; 25, *A* and *B*; and 26, *A* and *B*). The shadow is caused by calcification of the walls of the aneurysm at the periphery of the aneurysmal sac and a loss of continuity at the point where the sac communicates with the artery. The aneurysm usually occurs at or near the bifurcation of the renal artery into its two main intrarenal branches.[1]

Infarct. Renal infarctions may be classified as (a) arterial occlusion, (b) venous occlusion, and (c) traumatic infarction. There is a paucity of clinical reports of calcification following infarction.

Vascular Tumors. Calcification and ossification of vascular tumors of the renal arteries are rare pathologic phenomena.

F. LITHIASIS

The role of acute infection of the renal parenchyma, especially of the collecting tubules, in the production of microlithiasis and calcification of the basement membrane of the collecting tubules has been previously discussed.

Calcification and Ossification Associated with Renal Calculi. In cases of hydronephrosis and pyonephrosis accompanied by renal calculi, it is difficult, and often impossible, to determine whether the osseous process is primary (the result of metaplastic changes in the affected area) and independent of the renal calculi or secondary to irritation and infectious changes in the pelvic and calyceal mucosa induced by the renal calculi. Heterotopic bone formations in the pelvic and calyceal mucosa in cases of renal calculi are very rare findings.

Calcareous Incrustation of Pelvic and Calyceal Lining. Incrustation of the pelvic or calyceal lining by calcium salts may be visualized as a faint radiolucent shadow on the plain roentgenogram. The underlying pathology is a diffuse infection of the renal parenchyma and pelviocalyceal system by urea splitting organisms. The condition is more frequently unilateral, but may be bilateral.

CONCLUSIONS

1. Calcification of the kidney of the dystrophic nonmetastatic type is an infrequent, though not uncommon, pathologic and roentgenologic finding.

2. The dystrophic type of calcification is usually associated with a chronic destructive or degenerative lesion of the kidney, such as tuberculosis, pyonephrosis, abscess,

neoplasm, etc. This type of calcification is directly dependent upon the presence of local changes, such as hemorrhage, necrosis, or infection. The degree of calcification may be influenced by other factors, which include localized vascular changes (ischemia, infarction), altered phosphatase activity of the affected tissue, and alterations in the calcium and phosphorus content of the blood and tissue.

3. Ossification of the kidney and adrenal gland is a rare pathologic and roentgenologic finding and is usually associated with severe destructive diseases or neoplasm. The formation of bone in the lesions may be attributed to osseous metaplastic changes of the connective tissue in and about the pyelocalyceal epithelium, or to similar changes in the connective tissue of the stroma or walls of the affected area in cases of tumors, cysts, pyonephrosis, etc.

4. Dystrophic calcification of the kidney is readily discernible on plain roentgenograms. The calcified areas usually appear as one or more irregular patches or streaks scattered throughout the renal lesion.

5. In recent years an increasing number of cases of calcification in the adrenal area has been reported. These are most commonly associated with Addison's disease and, next in frequency, with an adrenal pseudocyst of the hemorrhagic type.

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All Sinai Hospital pathological reports are presented through the courtesy of Dr. T. Weinberg, Pathologist-in-Chief.

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THE DROMEDARY OR HUMPED LEFT KIDNEY*

LACK OF RELATIONSHIP TO RENAL ROTATION

By BENEDICT R. HARROW *and* JACK A. SLOANE
MIAMI, FLORIDA

FOR several years we have been cognizant of a peculiar humping of the left kidney which represents a normal variation. However, not until nephrography became more routine during intravenous urography could this entity be more clearly defined. In 1,000 consecutive excretory urographies performed in adult patients over a 2 year span in our office practice, this peculiarity has been noted in 23 patients. In only 2 other patients was the right kidney humped. The left renal deformity had also been observed on many urograms made at Jackson Memorial and Mercy Hospitals but the incidence was not tabulated.

LOCATION AND SIGNIFICANCE OF THE HUMP

In most of the cases the hump or bulge occurred in the mid-portion of the lateral border producing a triangular appearance to the left kidney. As discussed in one of our previous papers,[6] whenever a possible renal mass is noted on the plain roentgenogram, 30 cc. of 50 per cent sodium diatrizoate mixed with 20 cc. of 76 per cent methylglucamine diatrizoate is injected rapidly and roentgenograms are taken 20 and 30 seconds later. In most instances, an excellent nephrogram results. Figure 1*A* shows a nephrogram in a case of a humped, triangular left kidney. Figure 1*B* is the 10 minute intravenous urogram using compression. Well visualized is the typical elongation of the middle calyx, a common finding of this morphologic variant. On oblique roentgenograms the bulge disappears (Fig. 1 *C*). Another variant (Fig. 1, *D* and *E*) produces a rounded hump in the middle pole; still another triangular type has a more rounded form (Fig. 1*F*). Although the humping was not observed in the upper pole, a lower pole expansion is relatively common (Fig. 1, *G* and *H*) without elongation of the middle calyx. Thirty-five patients with an increased convexity of the lateral contour of the left kidney were also observed; however, the changes in these cases were not significant enough to be considered cyst or neoplasm and therefore were not classified as a true humped kidney. Increased convexity of the lateral border of the right kidney was rarely seen.

A search of the literature has failed to reveal publications on this deformity except for a recent paper by Friemann-Dahl;[3] yet, there is no question that this humping represents a definite normal entity. Persistence of fetal lobulations has not resulted in changes in the lateral contour of the kidneys with humping. Friemann-Dahl suggests that prolonged pressure from the spleen could result in this type of bulge or hump. A review of our material fails to reveal any relationship with the splenic shadow. Nevertheless, this theory remains plausible. In the embryo or infant, the spleen may prevent an expansion of the kidney anteriorly and laterally in one localized area with a resulting hump. Of course, as demonstrated by a review of Forde *et al.*,[2] enlarged spleens can displace the kidney downward, medially, occasionally cephalad, and may cause various types of kidney rotations but this acquired condition produces no bulging of the lateral contour of the kidney. Figure 1*I* is a nephrogram of a patient with a humping of the left kidney where there seems to be a relationship to the splenic shadow. In general, no such correlation could be proven.

In most of the cases, the hump occurred

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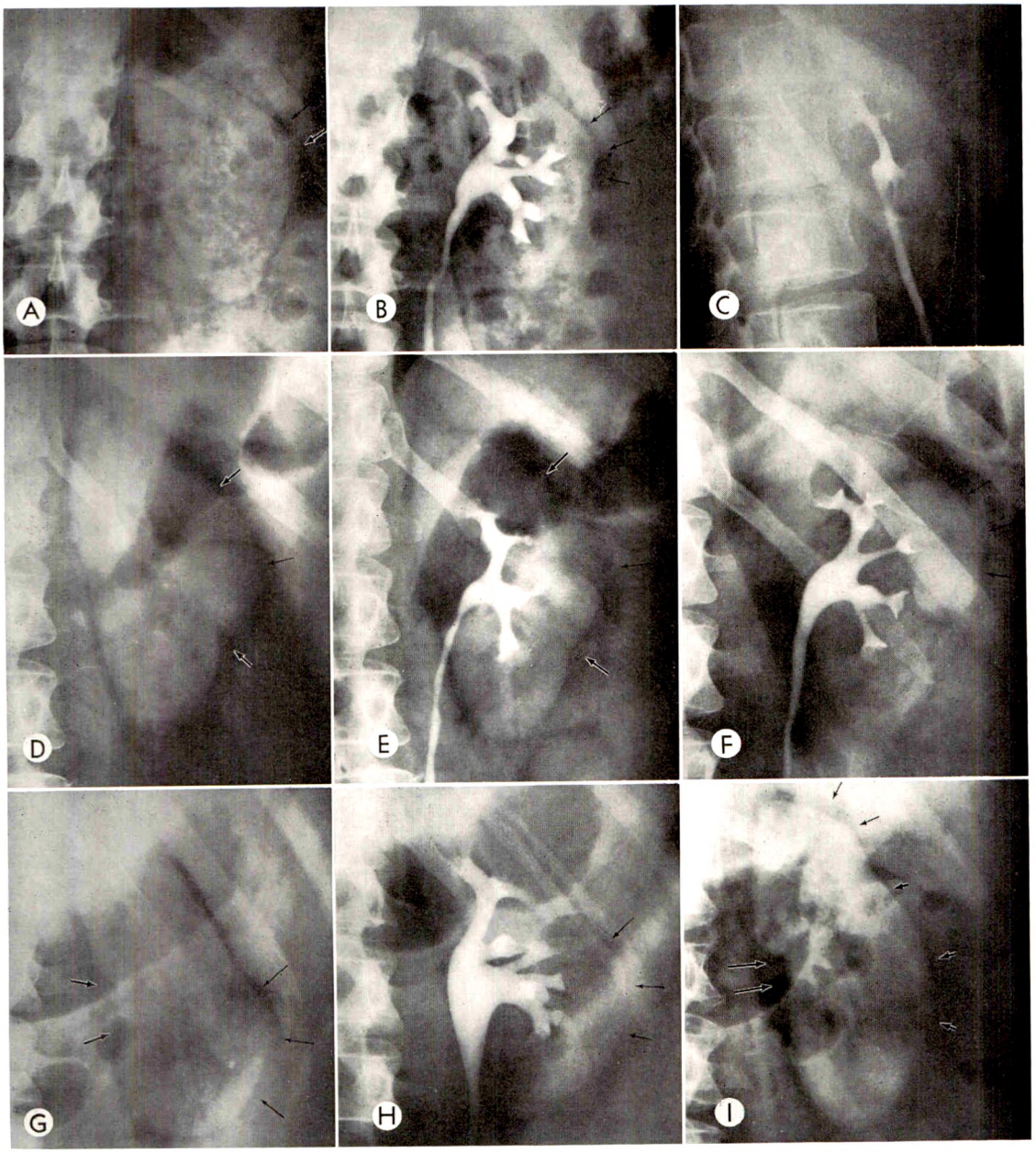


Fig. 1. (*A*) Arrows point to the humped portion of a typical triangular-shaped left kidney visualized on a nephrogram appearing during intravenous urography. (*B*) The 10 minute compression roentgenogram shows the normal but elongated middle calyx. (*C*) The bulge is not visualized on an oblique roentgenogram. (*D*) A well demarcated hump with peripheral depressions (arrows) is seen on this nephrogram. (*E*) The middle calyx of this left kidney is not elongated, differing from the typical triangular kidney. As in every case in this series, there is no localized hypoplasia with dysplastic calyces to account for the bulge. (*F*) A more rounded type of triangular kidney is visualized with only slight enlargement of the infundibulum of the middle calyx. (*G*) A pronounced humping of the lower pole is seen on the nephrogram appearing during intravenous urography (arrows). Arrows also point to a sharply-outlined notching of the hilus. (*H*) The 10 minute compression roentgenogram reveals only slight projection of the lower calyces into the hump. (*I*) Arrows point again to the notching seen on nephrograms of the left kidney more often than the right. White-lined arrows point to the lateral bulging. Black arrows show the notching of the spleen and the close relationship of the splenic shadow to the top of the renal hump.

as a coincidental finding in patients with other urinary tract pathology, and the left renal deformity was generally recognized as a normal embryologic variant in the morphology of the kidney. This type of roentgenographic finding of humped kidney can be confused with a cyst or neoplasm resulting in exploratory surgery. Knowledge of the condition can prevent this surgical procedure. In some instances the bulging may be present in a patient with gross or microscopic hematuria without other pathologic findings and, since an early neoplasm can possibly result in a similar finding, an exploratory operation may need to be performed in selected cases, especially when gross bleeding is identified from the left kidney. Follow-up examinations over a 2 year period on many of our 23 cases have failed to reveal any changes in the appearance of the kidney nor have the patients developed any left renal symptoms, thus ruling out any cyst or neoplasms in these humped kidneys. An appropriate designation for this morphology would be the term, dromedary left kidney. In the relatively few instances of right renal humping, there has been suspicion that an actual lesion existed, but further follow-up studies will be necessary.

Localized renal hypoplasia with or without calyceal dysplasia produces a relative bulging of the remainder of the kidney but this condition together with pelvic duplication was excluded from our series. Two other types of deformity in the renal outline that were not included in the 23 cases but can be compared with dromedary kidneys are illustrated by the following cases.

REPORT OF CASES

CASE I. J. G., Mercy Hospital admission #105746. A 48 year old white male with microscopic hematuria, discovered on a routine insurance examination, was found to have an enlarged, lobular pole of the left kidney. On the excretory urogram (Fig. 2*A*) the mass can be seen; Figure 2*B* is the retrograde pyelogram of this patient. He was explored through a left flank incision on July 20, 1959, and neither cysts nor neoplasm were found in the lower pole but rather an enlarged, lobulated lower section of the left kidney which extended toward the mid-line. One set of blood vessels entered the upper pole while a completely separate blood supply consisting of large arteries and veins coming from the aorta and inferior vena cava supplied the lower pole. At operation the lesion represented an obvious congenital deformity and therefore the lower pole was not split. A follow-up examination 2 years later revealed that the patient had remained well and a repeat intravenous urogram was identical to the original roentgenograms despite continuance of a microscopic hematuria.

CASE II. A. A., a 44 year old professional golfer was found to have a microscopic hematuria on several routine urinalyses. An intravenous urogram without compression revealed a lobulated lower pole of the right kidney which appeared to represent a cyst or neoplasm (Fig. 3*A*). During subsequent urography a nephrogram (Fig. 3*B*) again demonstrated the mass in the lower pole of this kidney. Next, on several roentgenograms, using compression with a balsa wood block, the right kidney was found to be rotated on its sagittal and transverse axes into a more normal position causing the disappearance of the apparent mass in the lower pole (Fig. 3*C*). Three seconds after release of compression (Fig. 3*D*), the kidney had almost returned to its usual position with a reappearance of the lobulation. Repeat urograms 3 years later showed identical findings.

The peculiar appearance of the right kidney in Case II was due to abnormal rotation without downward displacement with the lower pole being turned more medially and more anteriorly than the average kidney, resulting in a roentgenographic finding of shortening in the length of the kidney and a broadening of the lower segment. In general, this rotation on the transverse and anteroposterior (sagittal) axes has the same appearance that a mildly ptotic kidney assumes but in ptosis there is also caudal displacement. The bent stem appearance of many ptotic kidneys is a further accentuation of rotation on the transverse and sagittal axes.[4] Schematic drawings (Fig. 4, *A–D*) illustrate how the

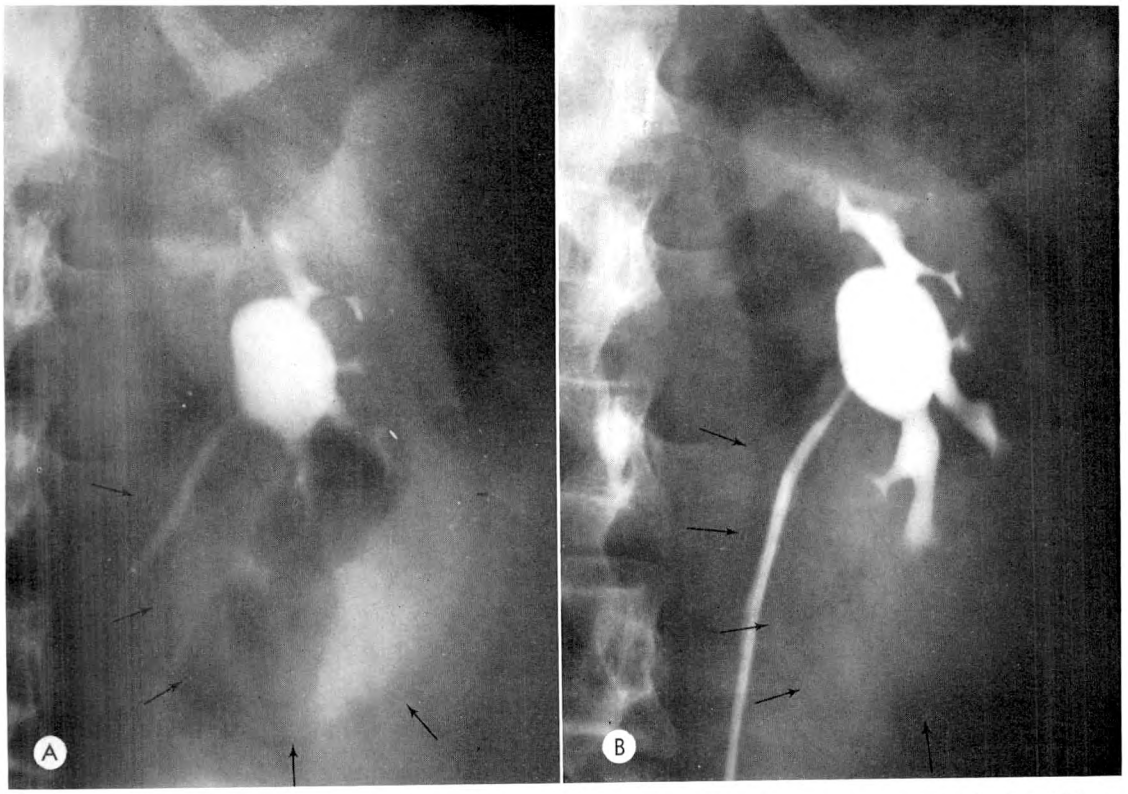


Fig. 2. Case 1. (*A*) An intravenous urogram shows an apparent lower pole mass (arrows) of the left kidney. (*B*) Retrograde pyelogram confirming the presence of the mass; however, at operation a congenital enlargement of the lower pole with a separate blood supply was found.

rotation mimics a space-occupying renal lesion. The common broadening of the lower pole of the right kidney due to rotation on the transverse axis should not be compared with a true humping.

DISCUSSION

In about 95 to 98 per cent of normal patients, the left kidney is more cephalad than the right or at the same level.[7,9] When the right kidney is more cephalad, the liver may overhang the lower pole of the kidney pushing the right kidney upwards as in many cases of hepatomegaly.[9] Gondos[5] has pointed out that the right kidney has a tendency to rotate on its longitudinal axis more than the left. This is caused by space limitations imposed by the liver, psoas muscle and fulcrum of the vascular pedicle. In longitudinal rotation the notching of the hilus tends to disappear and the convexity of the lateral contour decreases. In our series of 23 humped kidneys the notching was especially prominent on the left side (Fig. 1, *G* and *I*); however, the lack of rotation and the visualization of a broad left kidney does not explain the humping. Oblique roentgenograms of the right kidney (left side of the patient closer to the roentgenographic table) where the right kidney loses longitudinal rotation have not demonstrated any bulging. The term, increased longitudinal rotation as used above, reverses the usual embryologic terminology. This reverse is used deliberately because it implies that the kidney has already reached its full rotation embryologically (the posteriorly pointing calyces rotate almost 90 degrees to point laterally); however, space limitations do to some degree reverse the longitudinal rotation. Figure 4*D* illustrates the loss of hilar

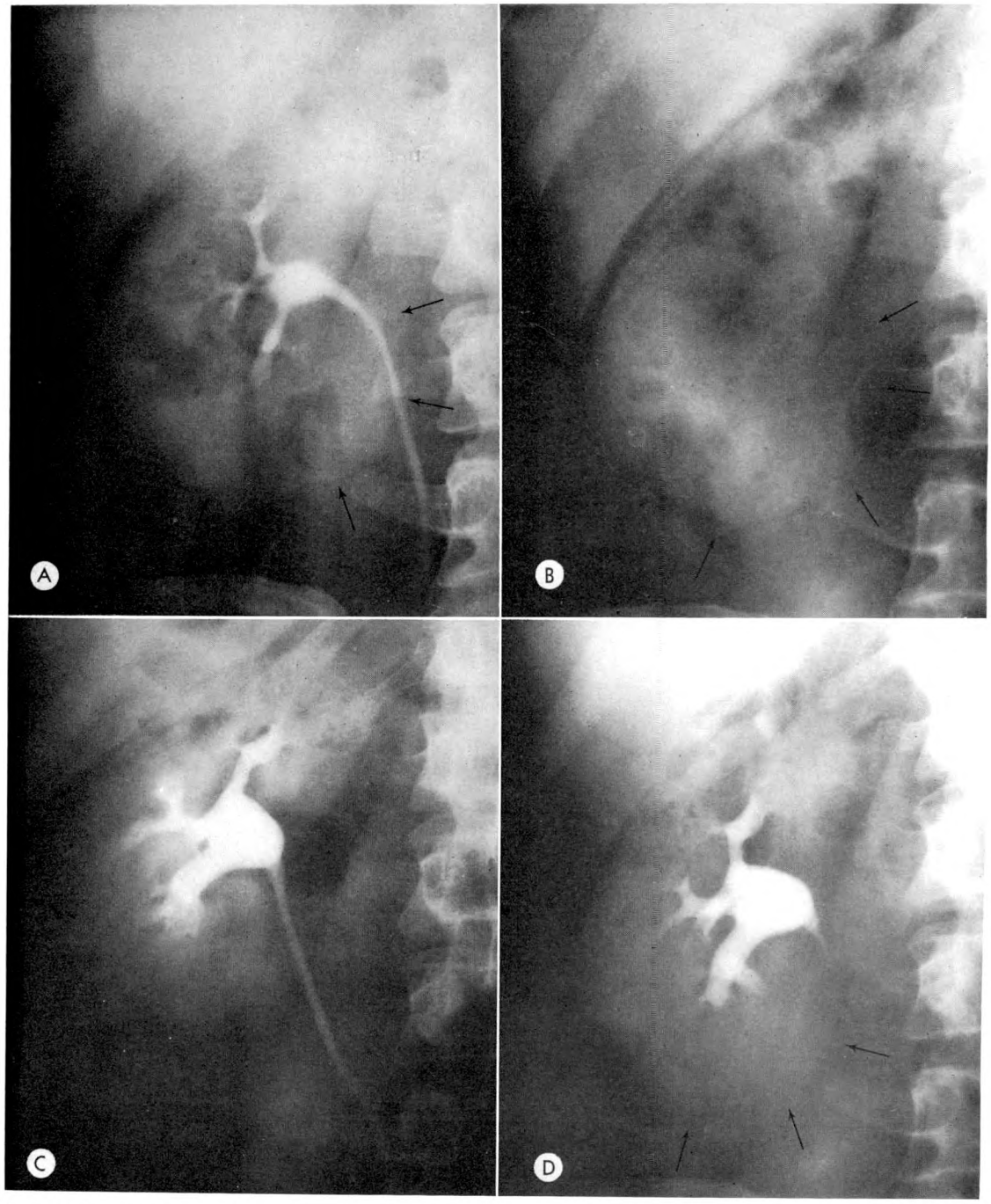


FIG. 3. Case 11. (*A*) An intravenous urogram without compression shows an apparent mass in the lower segment of the right kidney. Note the kidney is not ptotic but counterclockwise rotation on the sagittal axis is easily discerned. (*B*) During a subsequent intravenous urography, a nephrogram again demonstrates the apparent mass. (*C*) On the 10 minute roentgenogram with the balsa wood in place for compression, the calyces are filled to greater width than previously. Most important, however, is the easily-identified, clockwise rotation of the kidney on its sagittal axis. From the pressure of the wood block, the kidney also has rotated on the transverse axis, a deduction that is determined from the considerable increase in the length of the kidney. Rotation on both axes to a normal position has resulted in a disappearance of the apparent mass. (*D*) Three seconds after release of compression, the kidney has returned part way to its usual location. The mass again begins to appear due to counterclockwise rotation and to foreshortening from rotation on the transverse axis with the lower pole pointing anteriorly.

notching and the narrowing of the kidney with a decrease in lateral convexity which occurs in longitudinal rotation that counters the developmental rotation. Of course in the supine position the kidneys are normally rotated so that the calyces point somewhat more posteriorly, especially the right kidney as previously discussed. In the prone position the kidneys rotate; thus the calyces point in a true lateral direction depicting the kidneys in a broad projection on the anteroposterior roentgenogram.

Moëll[8] refers to several postmortem studies which showed the left kidney to be heavier and larger than the right kidney. In 150 apparently healthy soldiers, 25 women and 25 men without renal disease, he found by measurements of the renal outline on anteroposterior roentgenograms with a focus-film distance of 100 cm. that the left kidney was slightly wider than the right. Longitudinal rotation of the right kidney with a decrease in its width could possibly explain the difference except that he also found the left kidney to be longer than the right. The obvious hump seen in our 23 cases is too pronounced to be explained by differences in kidney size. In addition, in leukemic children, Besse *et al.*[1] could not substantiate Moëll's findings of differences between the right and left kidneys on roentgenograms or in their weights during postmortem examination, although he did corroborate the increase in size and weight of male kidneys.

The difference in longitudinal rotation between the left and right kidney is possibly one reason why the cortical index of Vuorinen *et al.*[10]

$$\frac{\text{(calyceal length} \times \text{width)}}{\text{(kidney length} \times \text{width)}}$$

may prove to be unreliable as a measure of renal function or capacity. Another reason is the frequent finding of other types of rotation, particularly along the transverse axis, as well as differences in the breadth of the kidney. Figure 5*A* shows a hydronephrotic kidney due to chronic bladder retention where the cortical index was .59, indicating a marked decrease in remaining kidney tissue; however, after one week of catheter drainage the kidney returned to normal with a cortical index of .44 (Figure 5*B*). Obviously, normal renal substance was present initially but could not be appreciated on an anteroposterior roentgenogram since the kidney had enlarged along its sagittal axis.

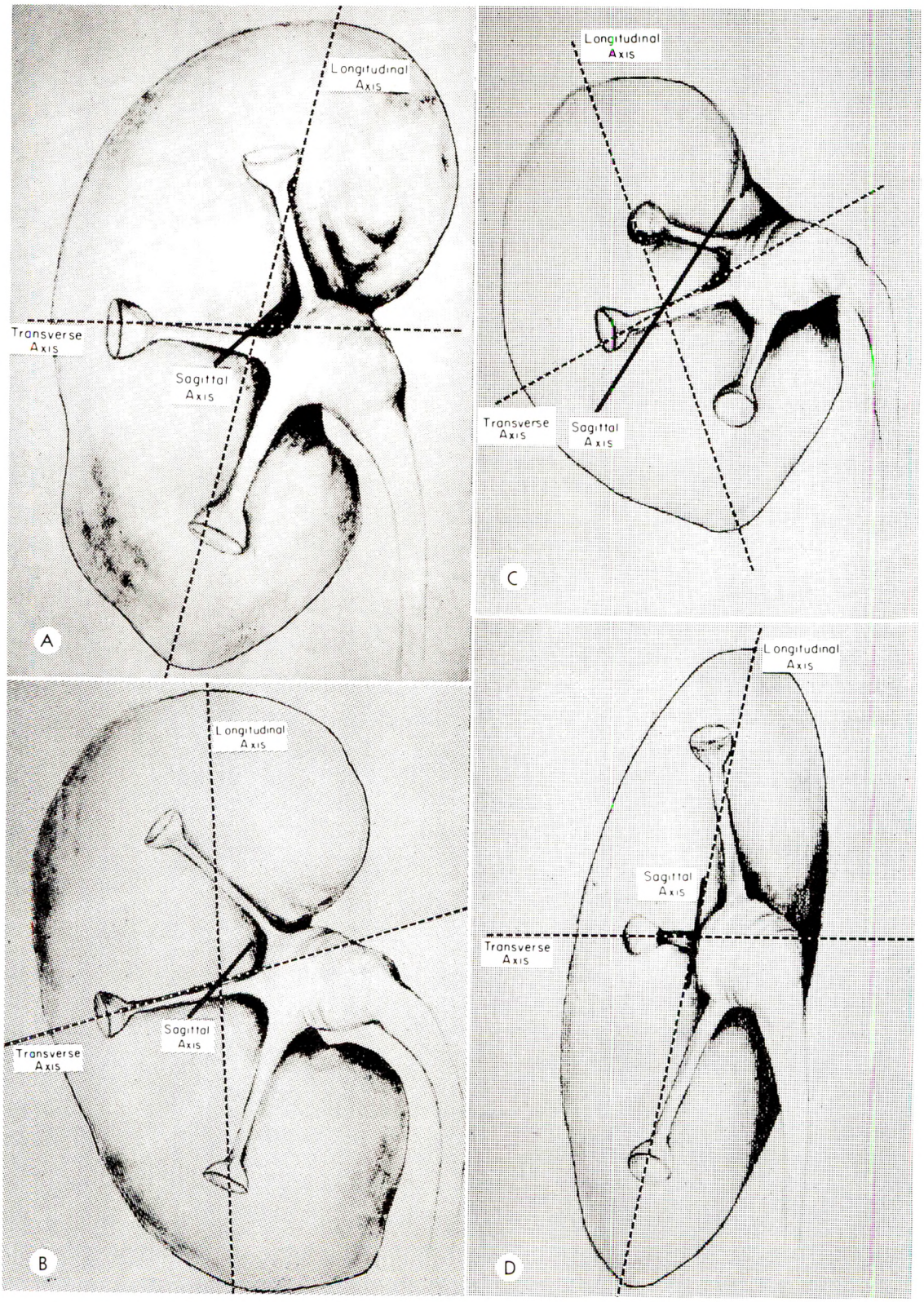
SUMMARY

A humped or dromedary left kidney is a common roentgenographic finding that has received little emphasis in the past. Recognition of this clinical entity may prevent an occasional exploratory operation for a suspected cyst or neoplasm. The hump cannot be explained by renal rotation or by an increase in the size of the left kidney over that of the right, and no correlation with the splenic outline appears to exist. In only a few cases was bulging seen along the lateral contour of the right kidney. Care should be taken not to confuse a true humping with a broadening of the entire lower pole of the right kidney due to foreshortening from rotation on the sagittal and transverse axes. The kidney with localized hypoplasia with or without calcyceal dysplasia must also be differentiated from the dromedary kidney.

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ADDENDUM

In an interesting new monograph on changes in kidney size due to acute renal failure, Moëll[1b] points out the necessary factors to be considered in comparing the length of kidneys on roentgenograms to the length of postmortem specimens. Of course, the lower pole of each kidney normally points slightly anteriorly due to slight rotation on the transverse axes. If the right kidney were normally rotated more on its transverse axis than the left, the increased length of the left kidney in anteroposterior views would be explained. It is difficult to answer this question at the present time even when lateral roentgenograms are taken. Decrease in detail and overlapping of kidneys prevent accurate


Longitudinal Axis
Transverse Axis
Sagittal Axis
A
Longitudinal Axis
Transverse Axis
Sagittal Axis
C
Longitudinal Axis
Sagittal Axis
Transverse Axis
B
Longitudinal Axis
Sagittal Axis
Transverse Axis
D

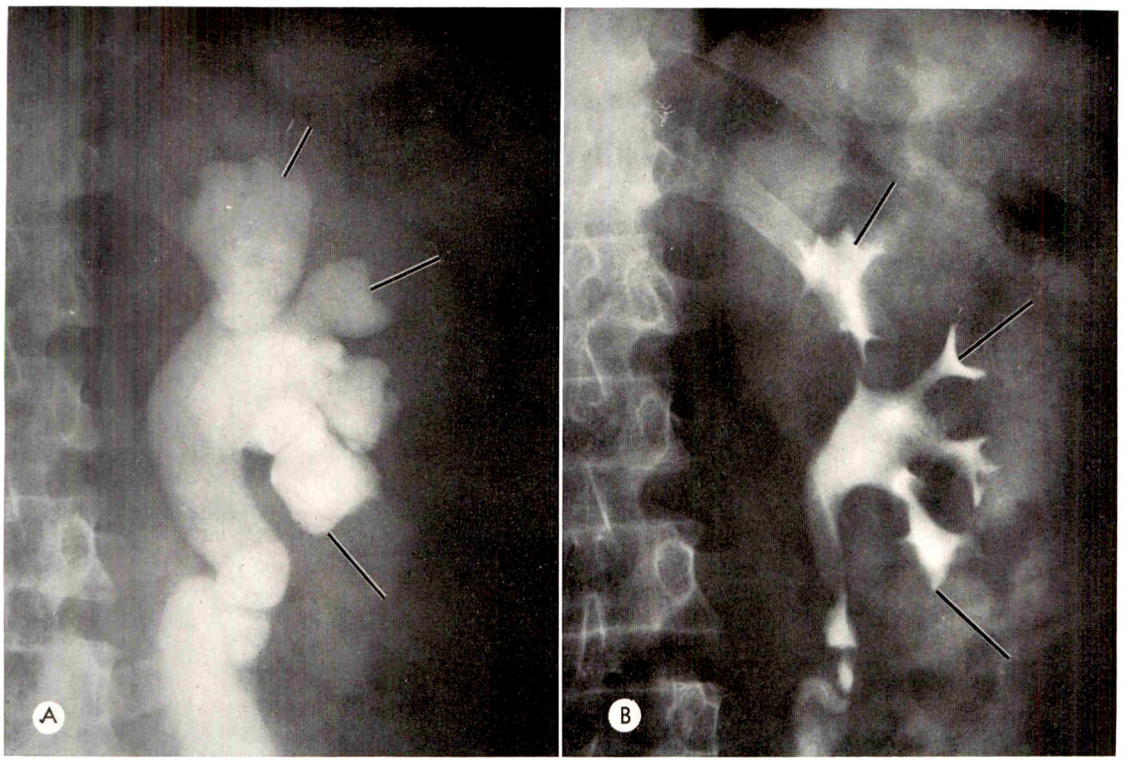


Fig. 5. (*A*) Considerable hydronephrosis is visualized on this intravenous pyelogram due to prolonged bladder retention from a benign prostatic fibroadenoma. (*B*) After one week of catheter drainage of the bladder, the calyces and pelvis returned to normal as seen on a repeat intravenous urogram. Lines drawn between the calyces and the renal outlines demonstrate how this distance has increased. The increase in the renal substance is only apparent, since these anteroposterior views do not take into consideration the breadth of the kidney.

appraisal. Moreover, evidence from postmortem specimens is great that the left kidney on the average is actually longer, wider and heavier than the right. Such knowledge of kidney size and position is important when comparing kidneys in hypertensive patients in attempts to diagnose renal artery blockage. However, the great variations not only in size but also in shape in normal patients even without rotation indicate that such comparisons are precarious in predicting actual unilateral renal pathology as a cause for hypertension.

Fig. 4. (*A*) A schematic drawing demonstrating the axes of a normally positioned kidney. Note the notching of the hilus. (*B*) Rotation counterclockwise on the sagittal axis is easily determined by the changes in direction of the upper and lower calyces. The notching remains and the length of the kidney is not decreased. Note that the positions of the longitudinal and transverse axes are changed but no rotation occurs along the latter axes. (*C*) The counterclockwise rotation is increased and now there is added rotation along the transverse axis with the lower pole pointing anteriorly. Note the decrease in length of the kidney causing foreshortening with the appearance of an apparent mass. The notching disappears and the sagittal axis elongates in this anteroposterior view. (*D*) During an isolated rotation on the longitudinal axis where the calyces begin to point more posteriorly instead of laterally, the hilar notching vanishes. The kidney width decreases with a decrease in the lateral convexity, although the length remains normal. For simplification, this rotation is considered a reverse of the normal embryologic rotation. It could also be called an incomplete rotation or a malrotated kidney from an embryologic viewpoint.

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MEDULLARY SPONGE KIDNEY AND POLYCYSTIC DISEASES OF THE KIDNEY: DISTINCT ENTITIES*

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AMONG the large group of polycystic conditions of the kidney it has recently been possible to distinguish yet another entity, namely, that usually known as medullary sponge kidney, from the Italian term *rene a spugna midollare* coined by the Italian authors, who were the first to define the condition as a specific disease.[4,11] The pathologic changes are restricted to the medullary zone and consist of cystic dilatation of the collecting tubules in the pyramids, in which multiple small calculi are often encountered. Although the disease was described some twenty years ago, it is only recently that it has come to be more generally reported in the English and American literature.[1,13,15,20,21] Some authors have made no distinction between this disease and other forms of polycystic kidney in infants and children.[9,10,18] It has therefore been considered of interest to examine the differences between these lesions.

MEDULLARY SPONGE KIDNEY

The following brief description of the medullary sponge kidney (Fig. 1, *A* and *B*; 2, *A* and *B*; and 3, *A* and *B*) is based on a personal series of 60 roentgenographically verified cases. Biopsy specimens from 19 of these were examined microscopically. For a detailed description of the disease the reader is referred to the monographs published by Ekström *et al.* in 1959[6] and Cacchi in 1960.[3]

The disease is generally discovered in middle age but it has also been found in young children and the aged. There are no primary symptoms, those experienced being due to calculus and infection. The renal function is usually good, although the concentration capacity may be slightly impaired. Except for frequent hypercalciuria the chemical laboratory tests usually give normal results.

The cystic changes can involve any number of papillae, in one or both kidneys.

The most striking pathologic feature of this disease is the presence of multiple small cystic cavities in the pyramids. These consist of both cysts and dilated collecting tubules. The cysts sometimes contain a clear jelly-like, often dark brown substance, and calculi and masses of calcific débris are commonly encountered. The microscopic picture varies considerably. In the larger cysts there is often stratified epithelium of the transitional type, occasionally of squamous or columnar nature. Some of the cysts with calculi have no epithelium. Sometimes they communicate with tubules or with one another, and in this case they can form a system of ducts, with a radial pattern of the normal ducts.

In the region of the pathologically altered papillae the normal ducts are often greatly reduced in number, if not entirely absent. A constant feature is a more or less severe inflammation, which, as a rule, is most pronounced in the immediate vicinity of the renal pelvis. The connective tissue within the affected regions is strikingly dense and cellular and differs distinctly from the normal interstitial connective tissue of the papillae. Rarely there are abnormal amounts of smooth muscle. The changes are restricted to the medulla, the cortex being entirely normal.

One characteristic roentgenographic feature is the presence of cavities of varying size and extent in the pyramids. These are sharply demarcated from the apparently normal tissue and vary in number from a few to many, as demonstrated by urog-

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FIG. 1. Right kidney. (*A*) The kidney is enlarged, containing calculi arranged in groups. The plain roentgenogram is typical of medullary sponge kidney. (*B*) Urogram. The calculi lie in round or oval cavities in the upper and lower pyramids.

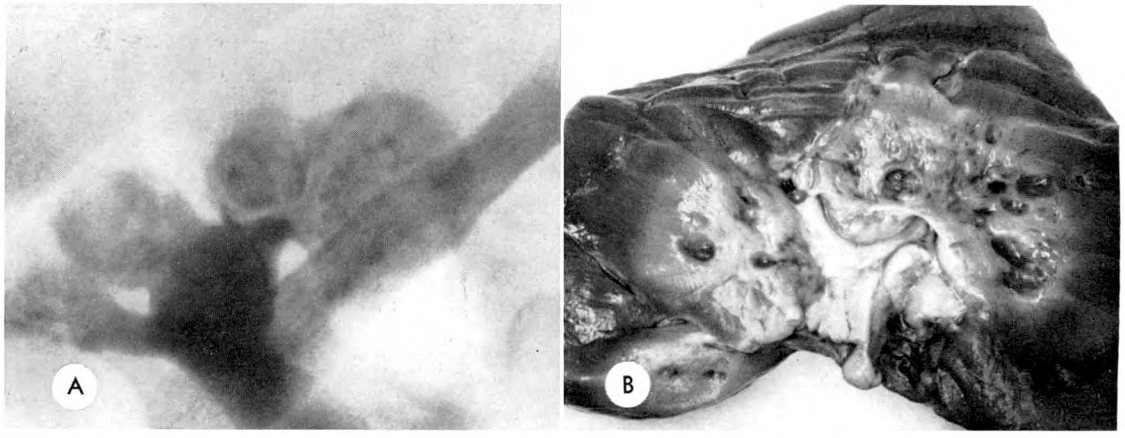


FIG. 2. Part of the right kidney; the superior pole is enlarged. (*A*) The urogram shows a typical medullary sponge kidney with slight enlargement of a few papillae. The pyramids contain cavities, most of them elongated and extending towards the base of the pyramids. When compression is applied to the ureter, some of the pyramids appear to be diffusely filled with contrast medium. Many small calculi are present in the cavities. (*B*) Resection specimen. The medulla is paler than normal. The presence of multiple cavities gives it a sponge-like appearance.

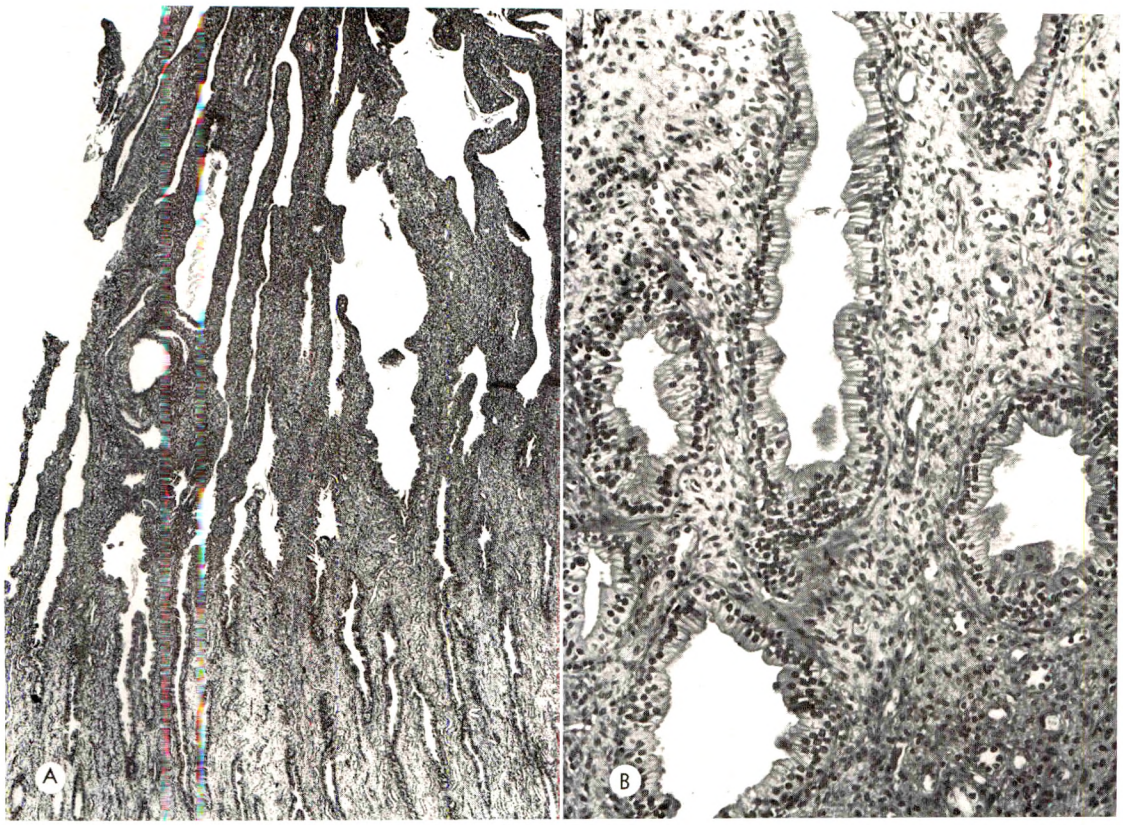


Fig. 3. (*A*) Medullary sponge kidney with irregularly dilated collecting tubules lined with high, pale columnar epithelium. Some collecting tubules of ordinary width are seen to become ectatic at the papillary tip (×30; van Gieson stain). (*B*) High power field shows the high columnar epithelium lining the ducts (×300; Hematoxylin eosin stain).

raphy which reveals diffuse filling of the pyramids, or parts of them. The cavities often contain calculi, their number likewise varying from one or two to several hundred. If the changes are present throughout the entire medulla of the kidney the organ can be enlarged. In cases where only part of the organ is involved there may be a local swelling. In mild cases no changes are visualized on the roentgenogram but histologically typical alterations are found. The roentgenographic appearances have been discussed in detail by, among others, Lindvall in 1959.[12]

Polycystic Diseases of the Kidney

The term polycystic disease of the kidney has sometimes been used for any kidney containing multiple cysts and at other times for certain specific types of lesions. Moreover, various authors have grouped these disorders differently and often the same terms have been employed to denote quite dissimilar cystic lesions. This lack of unanimity has resulted in considerable confusion in the literature. A further difficulty is the fact that the pathogenesis of most of the cystic conditions of the kidney is still unknown.

More recently, it has been possible to differentiate certain types of polycystic diseases, by virtue of their clinical and morphologic picture, from the medullary sponge kidney that can have multicystic changes. In this brief description of the polycystic diseases of the kidney which appear to have been confused with medullary sponge kidney the classification suggested by Allen[2] has been followed for the most part.

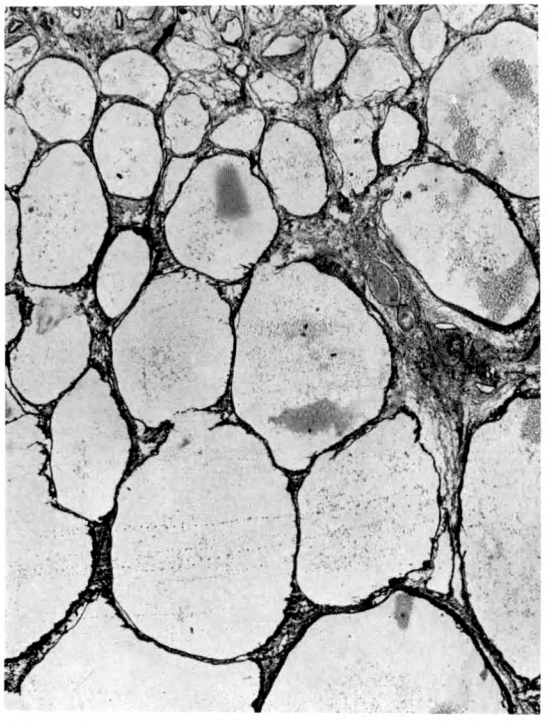


FIG. 4. Representative field of infantile polycystic kidney from stillborn infant. In the moderately enlarged kidneys, the cortex and medulla were completely transformed into spongy tissue composed of minute cysts. Note the normal amount of connective tissue surrounding the markedly dilated tubules. One small glomerulus is seen, there are no primitive ducts, ductules or cartilage (×70; Hematoxylin eosin stain).

A. ADULT POLYCYSTIC KIDNEY

This is one of the commonest polycystic renal diseases. It is familial and usually occurs bilaterally. It is often associated with cysts in other organs such as the liver and pancreas, and sometimes with intracranial aneurysm.[17] It is characterized by a slow insidious onset, with intermittent hematuria, albuminuria, hypertension, renal insufficiency in advanced stages and death in uremia. Most patients reach adulthood before the disease is discovered, but occasionally it can be diagnosed in the second decade of life. Examination discloses greatly enlarged kidneys with large, round cysts of varying size, which may contain blood-colored fluid. Sometimes there are a few calcifications in the cysts, the distribution of which almost always follows the direction from the cortex towards the medulla, showing no predilection for only one part of the kidney. The renal tissue between the cysts is normal.

The bilateral nature of the disease, the enlargement of the kidneys and the deformity of the calyces often result in a characteristic urographic appearance.

B. INFANTILE POLYCYSTIC KIDNEY

This form is fairly rare and is discovered at birth (Fig. 4). Death from uremia invariably occurs within the neonatal period. The disease is familial and bilateral.

The kidneys are usually enlarged, with normal configuration. The cysts are uniform in size and shape, and may be caused either by dilatation of the tubules or, rarely, by enlargement of the spaces within the Bowman capsules.[16] The changes occur throughout the kidney and give the cut surface a "honeycombed" appearance. The number of normal glomeruli varies and there is no increase in connective tissue. This disease is often associated with similar changes in other organs, especially the liver. Cysts in other viscera, for instance the pancreas or even the lungs, are rarely observed.

C. POLYCYSTIC DYSPLASIA OF THE KIDNEY

There are various grades of renal dysplasia,[19] some of them with cystic changes, which can constitute the dominating feature. This type of dysplasia is the most frequent variety of polycystic diseases of the kidney in childhood (Fig. 5). The condition is usually unilateral and is not believed to be familial.[14]

The clinical manifestations are of extreme variation. If the changes are bilateral, the disease is frequently fatal early in life.[16] Subjects with unilateral polycystic dysplasia not infrequently can reach adult age and have few clinical symptoms, unless the condition is complicated by pyelonephritis.[7,8]

There is a variation in the size of the kidneys, which may be enlarged or very small, and are often irregular in shape. The cysts, likewise, vary in size and shape and in severe cases may resemble a "cluster of grapes." The cysts occur in both the cortex and the medulla and they may contain precipitated calcium salts. There is an increase in the renal connective tissue and a reduction in the number of nephrons, which are composed of primitive elements. Frequently only a few glomeruli are seen and these are hyalinized with no definite orientation with respect to the tubular elements or calyces. Cartilage is sometimes present in the stroma.

The roentgenographic appearance of renal dysplasia has been described by Ekström,[5] who, however, seemingly had not observed any characteristic polycystic cases. As far as we are aware, the literature provides no detailed roentgenographic description of polycystic dysplasia.

CONCLUSION

It may be said that medullary sponge kidney is, from the clinical, roentgenologic and morphologic aspects, a well defined entity, which differs from the other three forms of polycystic changes in the kidneys (adult and infantile polycystic kidney and polycystic dysplasia) with which it has been confused. Since the cysts in the medullary sponge kidney, unlike those in the other three diseases, occur only in the medulla, it may be assumed that there is also a difference in the mode of occurrence of the congenital malformation which is considered to constitute the basis of all the diseases discussed here.

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Fig. 5. Unilateral polycystic dysplasia in a 13 year old boy. The increased connective tissue component forms concentric membranes around the primitive ducts. Cystic change is seen below the area of more normal renal tissue (×70; Hematoxylin eosin stain).

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COARCTATION OF THE RENAL ARTERY WITH "NOTCHING" OF THE URETER*

A ROENTGENOLOGIC SIGN OF UNILATERAL RENAL DISEASE AS A CAUSE OF HYPERTENSION

By MORDECAI HALPERN, M.D., *and* JOHN A. EVANS, M.D.
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HYPERTENSION of renal origin may be correctable by the use of contemporary surgical techniques. Renal arteriography by the percutaneous transfemoral catheter technique has been shown to be accurate, safe and easily performed. It has stimulated an enthusiasm for the investigation of the renal arteries in all patients in whom the possibility of renal artery disease exists as a cause of the hypertension. Our own use of this technique in the evaluation of the hypertensive patient[3] has uncovered 10 cases of occlusive renal artery disease in patients under the age of forty years.

Our interest is directed toward 2 of these patients both of whom showed an unusual contour deformity of the ureter on conventional pyelograms which is believed to be practically pathognomonic of the underlying arterial defect. The pathologic processes producing the renal artery stenosis in these 2 young individuals are subintimal fibromuscular hyperplasia and mural fibrosis. An adult female patient with severe hypertension demonstrated an identical ureteral deformity. A large segmental arteriosclerotic plaque in the abdominal aorta involved the orifice of the right renal artery and was the cause of the occlusive arterial disease.

REPORT OF CASES

CASE 1, #847852. In January 1960, J.H., a thirteen year old boy, was found by his physician to have cystitis and a blood pressure of 160/115. Following treatment with tetracycline and serpasil, he was admitted to The New York Hospital for study on March 7, 1960.

Physical examination revealed a blood pressure of 164/116 and grade 1 hypertensive changes in the fundi.

Pertinent laboratory studies were as follows: Na, 142; K, 4.5; phenolsulfonphthalein, 70 per cent total dye excretion with a normal curve; urea clearance 68 per cent first hour, 89 per cent second hour; regitine test normal; urine for catecholamines, upper limits of normal.

Intravenous pyelogram, retrograde pyelogram and cystoscopy showed a large bladder and a contour deformity of the left ureter (Fig. 1).

A renal arteriogram demonstrated the coarctation of the left renal artery with the appearance of a "jet" and a poststenotic dilata-

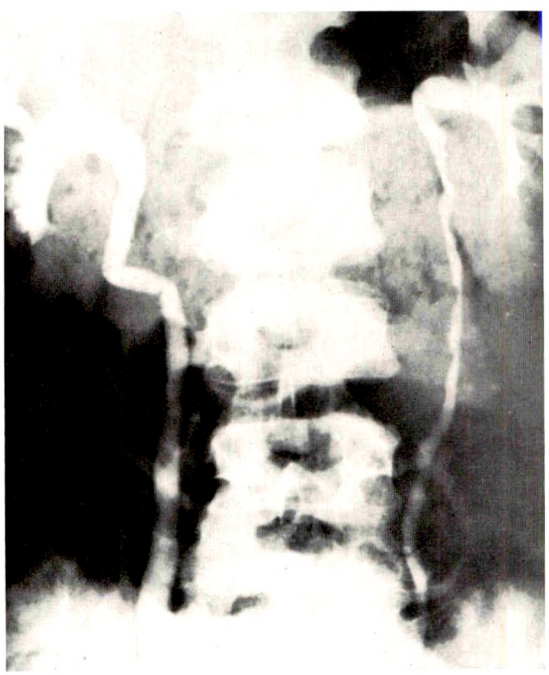


FIG. 1. Case 1. Pyelogram demonstrates numerous sharply defined, scalloped contour deformities of the left ureter.

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Aided by a Grant from the Whitehall Foundation.

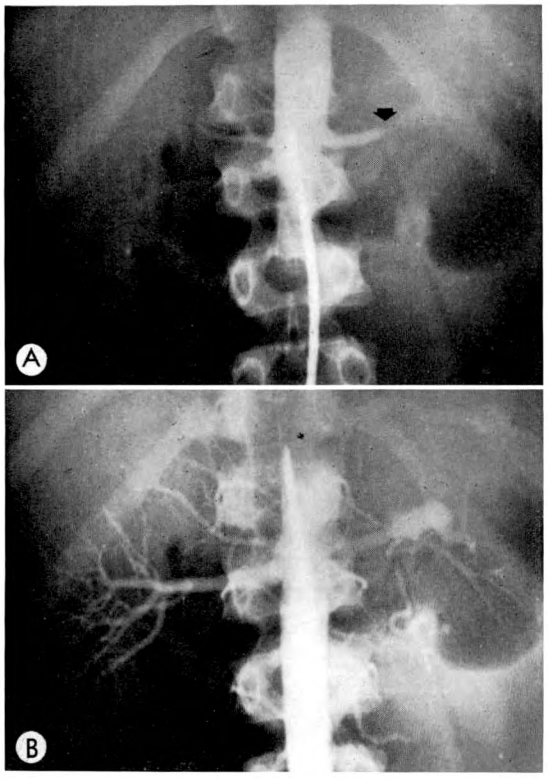


FIG. 2. Case I. Renal arteriograms show coarctation of the left renal artery with a "jet" sign (*A*) and poststenotic dilatation (*B*). *B* was made ½ second after *A*. The arterial collaterals are not well visualized.

tion (Fig. 2, *A* and *B*). Collateral circulation around the left ureter was evident.

A splenorenal arterial anastomosis was performed on March 21, 1960. The blood pressure returned to a level of 140/90 within two weeks and subsequently returned to normotensive levels which have been maintained to date.

CASE II, #865090. E.G., a sixteen year old female, was admitted to The New York Hospital on October 24, 1960 for an evaluation of hypertension.

One year before admission, a physician noted a blood pressure of 180/120 while the patient was being treated for an upper respiratory infection. She complained of bifrontal and bitemporal lobe headaches. There was no history of flank or abdominal pain or trauma and no urinary symptoms.

On admission the blood pressure was 205/110 and there was minimal arteriovenous nicking on fundoscopic examination. A grade I apical systolic murmur was present.

Laboratory studies were as follows: Na, 143; K, 3.6 and 3.4; Ca, 9.8. The regitine test was negative.

A hippuran renogram demonstrated hypovascularity of the right kidney.

The percutaneous transfemoral renal arteriogram showed the coarctation of the right renal artery with collateral vessels entwined around the ureter (Fig. 3). Figure 4 shows the scalloped contour deformities of the right ureter.

At exploration on November 2, 1960, the fragility of the wall of the artery made nephrectomy necessary (Fig. 5). The blood pressure was 130/90 on November 11 and 120/80 by November 20, 1960, which level has been maintained to the present time.

CASE III, #789046. L.H., a forty-nine year old female, was first seen in 1955 with a chief complaint of occipital headaches. Systolic blood pressure was 260. The first New York Hospital admission was in 1958 at which time the blood pressure was 250/130. Arteriolar narrowing, arteriovenous nicking and numerous exudates in the fundi were noted. The blood urea nitrogen was 26 mg. per cent, the urea clearance was 46.5 per cent in the first hour and 48.1 per cent the second hour.

An excretory urogram showed normal bilateral function and the presence of a calcified arteriosclerotic plaque in the aorta at the L1 level. The regitine test was negative. An I^{131} renogram demonstrated decreased function bilaterally both in the excretory and secretory phases. The patient was discharged from the hospital on a regimen of antihypertensive drug

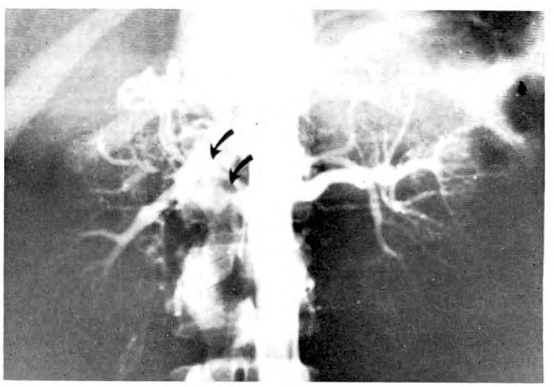


FIG. 3. Case II. Renal arteriogram demonstrates occlusion of the proximal portion of the right renal artery and an extensive plexus of vessels in the region of the renal hilus and along the course of the right ureter.

therapy. The blood pressure remained persistently high, ranging between 230–260 systolic and 120–130 diastolic.

The patient was readmitted to the hospital in December, 1960. Roentgenograms (Fig. 6 and 7, *A* and *B*) showing some of the findings are reproduced. The unusual findings of the percutaneous transfemoral renal arteriogram are the subject of a separate report.[4]

DISCUSSION

In the study of hypertension there has been a diligent search for screening methods and for valid all-encompassing indications for arteriography. However, it is pertinent to refer to intravenous and retrograde pyelography. As early as 1943 Shrader *et al.*[10] compared 100 normal pyelograms with 100 pyelograms in hypertensive patients but were unable to find any significant statistical evidence of difference in the details of apparent function or morphology. In the past few years a 1 to 2 cm. difference in renal size has been widely used as a criterion for arteriogrpahy in the hypertensive patient.[9] Excretory urography as a parameter of renal function has been investigated and function is also being

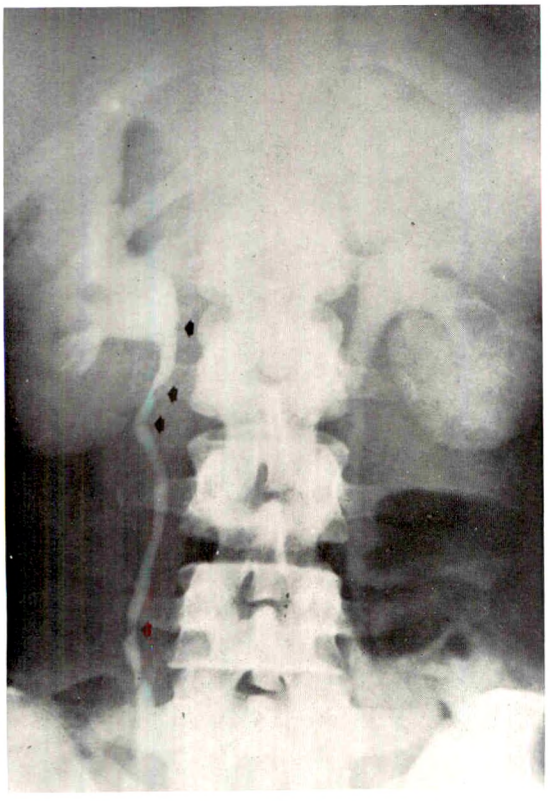


Fig. 4. Case II. Conventional pyelogram demonstrates the scalloped contour deformities of the right ureter.

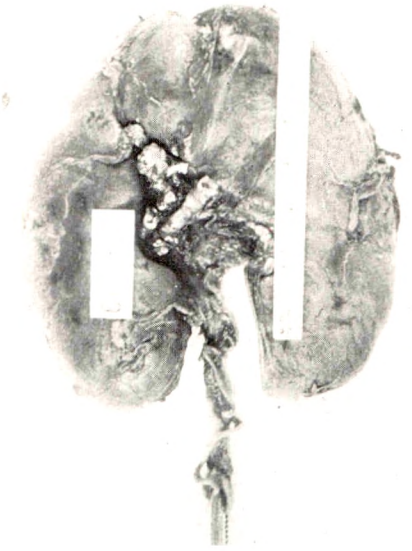


Fig. 5. Case II. Surgical nephrectomy specimen shows the dilated ureteral anastomotic vessels.

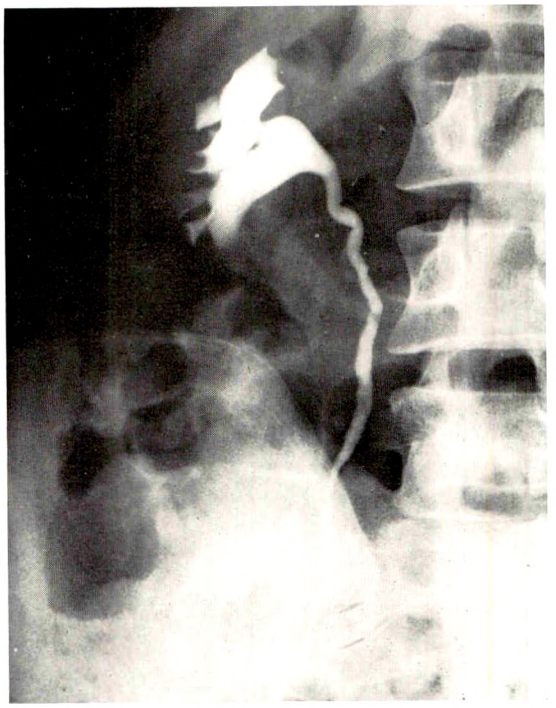


Fig. 6. Case III. Pyelogram demonstrates the scalloped contour deformities of the right ureter.

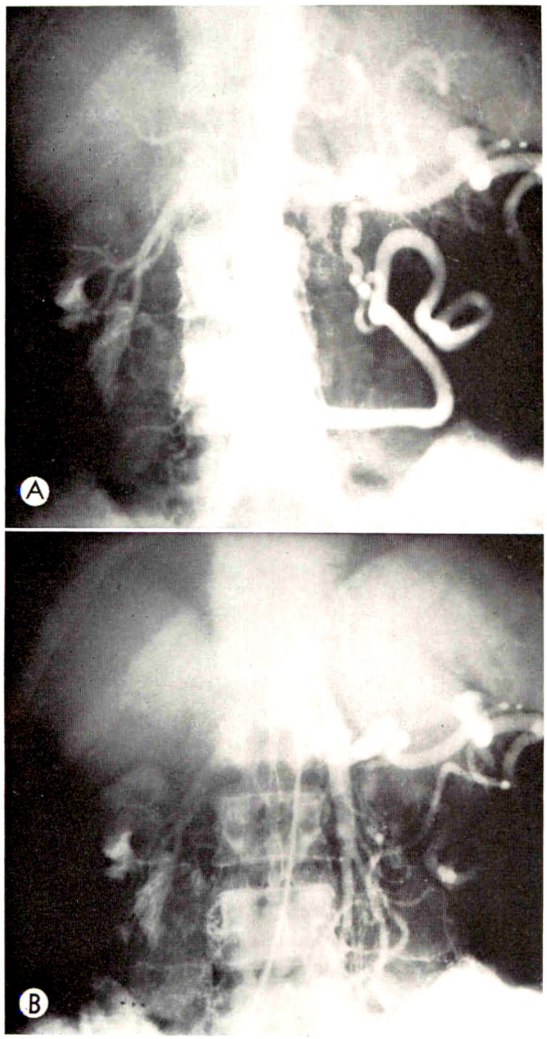


Fig. 7. Case III. (*A*) Renal arteriogram shows non-opacification of the aorta in the region of the right renal artery orifice and occlusion of the proximal portion of this artery. (*B*) The extensive arterial plexus is seen along the path of the right ureter at the L_{3-4} level.

studied by isotope renograms and fractional excretory urography.

It is the purpose of this paper to call attention to an interesting and uncommon ureteral defect associated with segmental stenosis of a renal artery. The scalloped contour defects of the ureters shown in Figures 1, 4 and 6 represent an unusual finding which has been rarely mentioned in the medical literature. It is felt that coarctation of the renal artery is, in all probability, a congenital lesion, and that an attempt at collateral blood supply to the kidney is made through the long ureteral arteries arising from the aorta which anastomose with branches from the renal, testicular or ovarian arteries in the normal person. Secondary branches of the ureteric arteries form a plexus around the ureter. This produces dilatation and tortuosity of the involved arteries with the resultant "notching" of the ureter appearing on arteriograms of our cases (Fig. 3 and 7, *A* and *B*) and confirmed by the surgical specimen of Case II (Fig. 5).

There can be little doubt that such a finding on conventional urographic studies in a hypertensive patient represents a convincing indication for renal arteriography. It is interesting that in 1957 Isaac *et al.*[6] noted identical ureteral defects in 2 hypertensive patients described as having congenital cirsoid anomalies of the renal arteries and concluded that such a finding indicated the necessity for arteriography.

The literature contains a few scattered reports of calyceal compression deformities, with or without associated calcification, caused by aneurysms of the renal artery.[1,5,7] In one instance an arteriovenous aneurysm was present.[8] There appears to be a single report in the literature of filling

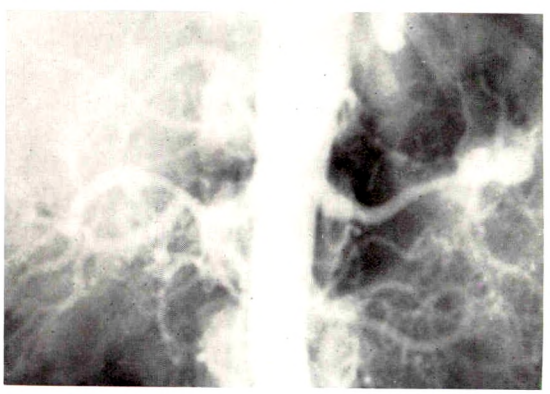


Fig. 8. Bilateral renal artery stenosis in a twenty-two year old female. The renal arteriogram demonstrates numerous small arteries in the region of the renal hili. Note the narrowing and "beading" of the distal portion of the right renal artery and its branches. Note also the coarcted segment of the left renal artery with two poststenotic aneurysms.

defects of the ureter caused by a varicose ureteral vein.[2] Retroperitoneal fibrosis has been reported[11] as a cause of varicosities surrounding the ureter to produce contour defects along the pelvis and ureter.

The differential diagnosis of "notching" of the ureter would include such conditions as ureteritis cystica, chronic ureteritis, ureteral tumors, paraortic lymphadenopathy, retroperitoneal fibrosis and varicosities surrounding the ureter. Only the last two conditions would closely simulate the oblique, entwining scalloped defect under consideration. The correct diagnosis would readily be established by means of history and arteriography.

It must not be assumed that this ureteral deformity will be manifest in all cases with severe stenotic lesions of the renal artery. Figure 8 is a percutaneous transfemoral renal arteriogram of a twenty-two year old female with bilateral renal artery lesions. There is evidence of some degree of collateral blood supply; however, excretory urograms did not demonstrate the anticipated ureteral defects.

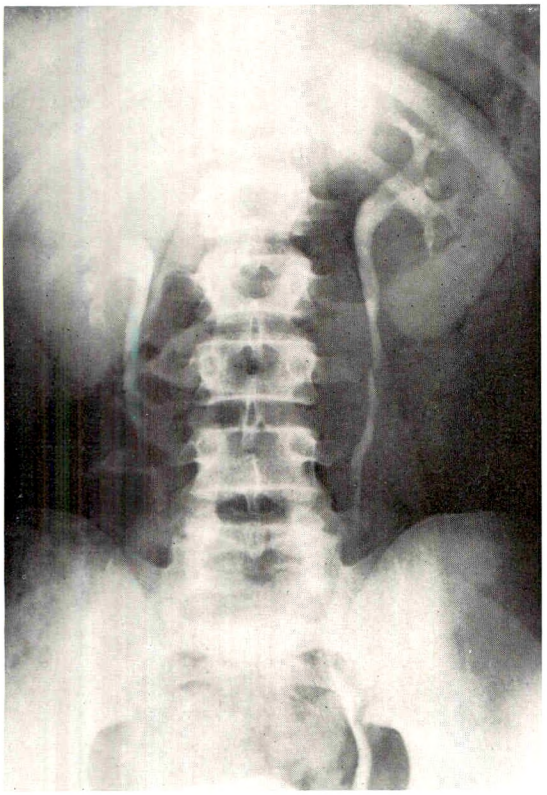


Fig. 9. Ureteral varicosities in a twenty year old male. The pyelogram demonstrates many "notched" contour deformities of the upper margin of the left renal pelvis and the ureter.

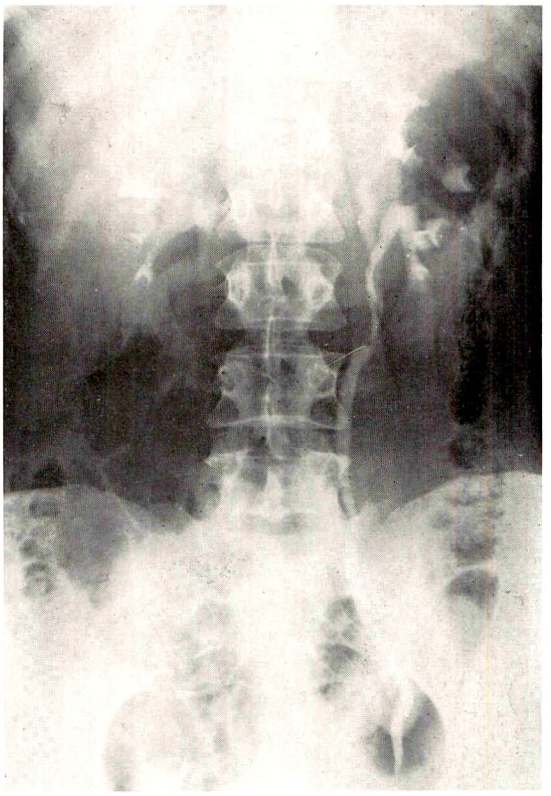


Fig. 10. Pyelogram shows undulations of the left ureter due to peristalsis and ureteral tortuosity.

The rare demonstration of ureteral notching secondary to venous varicosities is seen in Figure 9. This pyelogram was obtained in the course of investigation of sporadic idiopathic hematuria in a twenty year old male and the diagnosis was confirmed at exploratory surgery. The findings on the excretory urogram are indistinguishable from those of patients with arterial collateral circulation.

Finally, differential diagnosis must include ureteral peristalsis and tortuosity. The forty-five year old female patient shown in Figure 10 did have a recent onset of hypertension and also had a minimal stenotic lesion of the left renal artery. Col-

lateral circulation was not visualized on the renal arteriogram nor was it seen at the time of surgery. The undulating borders of this ureter may mimic notching but the sharp scalloped defects of true ureteral notching were not present.

CONCLUSION

We have reported 2 cases of congenital "coarctation" of the renal artery in patients thirteen and sixteen years of age and a case of arteriosclerotic occlusive renal artery disease associated with "notching" of the ureter.

"Notching" of the ureter is presented as a new roentgenographic sign of stenosis of the renal artery and as an indication for renal arteriography.

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ADDENDUM

Since this paper was submitted for publication, 2 additional patients with notching of the ureter and renal artery stenosis, a 22 year old male and a 16 year old male, have been studied. Dr. A. Bajek, Summit, New Jersey, was also kind enough to submit the urogram and arteriogram of a 14 year old girl with identical findings. In addition, Thomas, R. G., and Levin, N. W. reported a case of unilateral renal arterial stenosis and ureteral scalloping (*Brit. J. Radiol.*, 1961, *34*, 438–440).

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Vol. 88, No. 1

AN AUTOMATIC TIMER FOR USE IN RENAL ANGIOGRAPHY

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THE essential requirement for roentgenograms of consistent maximum clarity and quality in renal angiography is exposure at the instant of maximum filling of the renal vessels. Ordinarily, angiography is performed with a hand syringe and a verbal order or understanding that the x-ray technician choose the moment of exposure.[2] Multiple and quick changing cassettes along with automatic injectors are also used to obtain better results. However, these techniques involve expensive equipment, or the generation of injection pressures up to 800 mm. of Hg, when 20 cc. of contrast medium is injected through a No. 16 needle.[3]

The translumbar technique for renal angiography used at the Massachusetts General Hospital has been described in detail.[1] Briefly, this consists of injections of not more than 10 cc. of 50 per cent sodium diatrizoate† through a No. 17 thin-walled needle by means of a 10 cc. Luer-Lok control syringe. Control of the moment of roentgen exposure was dependent on verbal command or the reflexes of the technician and delayed roentgenograms were obtained by rough mental timing after the injection had been completed. Elimination of these variables has been achieved with a simple automatic timer to be described.

APPARATUS

A 10 cc. Sana-Lok control syringe is fitted with 2 terminals for a 6 volt circuit. One is screwed into the handle of the control ring and the other which is in the form of a sliding washer is fitted onto the thin metal plunger of the syringe (Fig. 1). This latter terminal rests on a nylon or hard rubber bushing, which allows it to slide on pressure but insulates it from the plunger. At the end of the injection this sliding terminal contacts the metal cap on the top of the syringe barrel, and thus it closes the 6 volt circuit, actuating the timer for either instantaneous or delayed exposure by the roentgen ray unit.

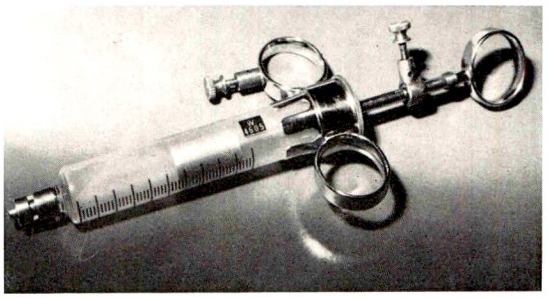


FIG. 1. The control syringe showing the 2 terminals.

The terminals on the syringe are made of brass, with small holes to accommodate male plugs which are screwed in place. These plugs are part of a thin flexible twin teflon wire cable, which connects the syringe to the timer box. This allows free movement whenever handling the syringe. The timer (Fig. 2) is powered by line current. A self-contained transformer supplies the 6 volts for the syringe circuit. The timer unit is graduated from instantaneous exposure to a maximal 12 second delay adjustable in 1/8th second divisions. The cycle is started through a 6 volt/110 volt relay with a momentary starting impulse, which initiates the cycle for instantaneous or delayed action. When this cycle is completed, the usual 110 volt exposure circuit of the roentgen ray unit is closed. This latter circuit is held in the closed position after cycling, until the timer is reset by a push-button. A green pilot light indicates that the unit is switched on and is being supplied

† Supplied as Hypaque, Winthrop Laboratories, New York City.

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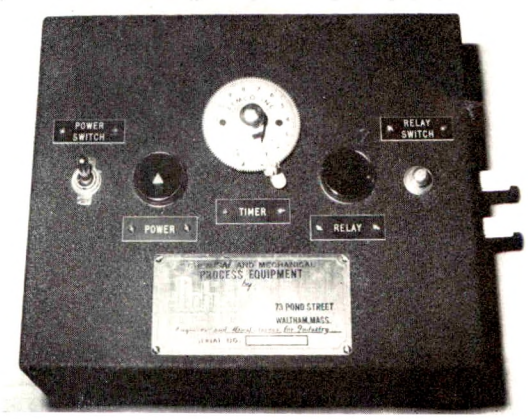


FIG. 2. The timer unit.

with power. A red pilot light glows when the unit has been fired and remains on until it is reset for the next exposure. The unit is explosion proof, made of strong metal, and easy to operate. There are 2 hooks on the back of the timer so that it may be attached to the side of the operating table for convenient visible control by the operator.

TECHNIQUE

The technique employed for renal angiography is standardized, being described in a previous publication.[1] To use the timer, it is first attached to the operating table, then switched on. Contacts are checked to ensure proper working order, the teflon cable is attached to the timer and then to the syringe. With the aortic needle in position, the roentgen ray unit is switched on and the injection begun. As soon as the terminals meet at the end of the injection, the timer is actuated, and the exposure is made automatically at a consistent, accurate and known moment. By moving the terminal, which slides on the plunger, downwards beforehand, it is possible to complete the exposure during the injection of the contrast medium. Thus, quantities smaller than 10 cc. can be injected and the terminal on the plunger adjusted accordingly.

RESULTS

This device has been used over the last year to achieve improvement in quality and clarity of renal angiograms by means of a known accurate exposure time. An injection of 10 cc. of 50 per cent sodium diatrizoate, using the equipment and technique described, takes an average time of 0.65 seconds and generates a transient peak pressure of 250–300 mm. Hg at the tip of the needle. Instantaneous exposures are obtained for the first test shot when diluted medium is used to check needle position. When the needle has been placed within 1 to 2 cm. of the renal arteries, the best quality exposures have been obtained with the timer set at a delay of 0.5 seconds. Obstructive lesions of the renal arteries may need a delay of 1 to 3 seconds. It is not possible to establish absolute time factors, as the position of the renal arteries in relation to the needle tip, the rate of aortic blood flow, and other anatomic and pathologic variants preclude standardization. However, as an aid to better roentgenographic quality, with more accurate diagnosis of minor abnormalities it does permit control of what has been a very variable factor. There were some initial technical problems relative to the early design of the timer circuit. These have been corrected and the unit can be used accurately after a short period of familiarization.

DISCUSSION

The simple timing device described is an aid to better renal angiographic technique, and in no way replaces the need for a trained physician performing the procedure. Excellent angiograms can be obtained without it, particularly with a constant and permanent staff; but, when used as suggested, roentgenograms of consistently improved quality and clarity will result. The same essential mechanism can be adapted to examinations of other vessels and other viscera, where injection of contrast medium is necessary.

SUMMARY

A simple automatic syringe timer mechanism is used to activate a roentgen ray

unit either instantaneously, or after a controlled delayed period. This device allows the operator to obtain roentgenograms at the optimal moment of delineation of the desired part, during or after injection of contrast media.

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Appreciation is acknowledged to Mr. Roy McDowell and Mr. Oliver Merril for metal work and electrical advice respectively. The unit is obtainable from Artisan Industries, Pond Street, Waltham, Massachusetts.

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AN EXPERIMENTAL STUDY OF THE MOTTLE PRODUCED BY X-RAY INTENSIFYING SCREENS*

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PERHAPS the best way to introduce our subject is to describe a simple experiment which can be performed in any radiographic laboratory. An attempt to explain the phenomenon observed in this experiment led to a series of additional experiments the procedures and results of which we shall describe. We believe that the results of the various experiments carried out in our Laboratories lead to a unique explanation.

First, we shall describe a basic experiment[6] which illustrates the phenomenon. A cassette equipped with intensifying screens is loaded with a screen-type radiographic film. Exposure conditions are chosen so that the kilovoltage is within the range commonly used for diagnostic radiography and a distance of 5 or 6 feet is used to ensure reasonable uniformity of the roentgen rays over the cassette area. Enough filtration is added at the tube port so that the exposure will be within the range of everyday practice. The most essential requirement for the success of this experiment is that there shall be good contact between the film and the screens. Briefly, then, the conditions for the exposure are similar to those which might be used in a diagnostic procedure except that additional filtration at the tube provides absorption which would normally be furnished by the patient's body.

The second part of the experiment is similar to that just outlined with the important exception that poor contact between film and screens is deliberately introduced. In fact, the screens should be spaced one-sixteenth of an inch or more from each side of the film. The simplest way to do this is to cut two sheets of thick cardboard to fit inside a cassette. (It may be necessary to use an old cassette and remove the hinges so as to accommodate the extra thickness.) A hole about 2 inches in diameter is cut in each of the cardboards and the holes are located so as to be in approximate register. With the intensifying screens in their normal position, the cassette is loaded with a sheet of screen-type x-ray film with the cardboards on both sides. Thus, a 2-inch-diameter area of the film will receive screen light which is quite diffuse. Only the minimum pressure should be used in closing the cassette so as to avoid distorting the screens which otherwise might make partial contact through the holes in the cardboards.

The two films should be processed together and any other precautions taken to make certain that they will have practically the same density. For best results, the density should be between 0.6 and 1.0. The film which is spaced between the screens will not receive appreciably less exposure in its central area than if it were in contact because the spacing distance is small compared with the dimensions of the exposed area.

Comparison of the two processed films will show a striking difference. The density of the spaced film will appear quite uniform to the unaided eye but that of the other will show an irregular pattern of small density variations. Since the film can only record what it "sees," there must be an unevenness in the distribution of light at the surface of the screen. This pattern is not recorded on the spaced film because, with the fluorescent light spreading out from each element of the screen, the small variations in brightness of the screen are diffused

* From the Research Laboratories, Eastman Kodak Company, Rochester, New York (Communication No. 2243). The Eastman Kodak Company does not manufacture x-ray intensifying screens in the United States.

or "smoothed out" at the film plane. The irregularity in density of the film which was in contact with the screens we have called "screen mottle" (Fig. 1).

Does film graininess play a part in this phenomenon? Probably, but it is slight. An uneven density variation can be seen in the spaced film at low magnification. This film graininess is caused by the random clumping of the minute particles of silver which have been reduced from silver bromide during development. The silver grains can only be seen with the aid of a microscope at fairly high magnification. Thus, the unevenness in density due to uneven light emission from the screen is of a higher order of magnitude than film graininess which has its origin within the radiographic emulsion.

It is natural to assume that the uneven density from a screen exposure in which there is good screen–film contact arises from some built-in characteristic of the phosphor coating.[2,11] For example, suppose there were a random clumping of the minute crystals of the phosphor before the coating solidified. This would surely affect its optical properties, and hence its emission, in an irregular manner. If screen mottle is caused principally by a built-in structure of the phosphor coating, *exactly* the same pattern should be recorded on the film for every exposure; but if the pattern is of a random nature, no two will be alike. Accordingly, two successive x-ray exposures of the same area of a single commercial intensifying screen were made on a single emulsion of Kodak Blue Brand Medical X-ray Film. To facilitate analysis, both were photographically enlarged 4 diameters and superposed for viewing. The patterns could not be brought into register, thus indicating that the two patterns were not alike. In fact, no moiré pattern* could be produced. We concluded from this test that the pattern had its origin in the statistical distribution of photons absorbed in the screen rather than from the screen's structure. The former pattern we call "quantum mottle," the latter "structure mottle," both together comprising "screen mottle."

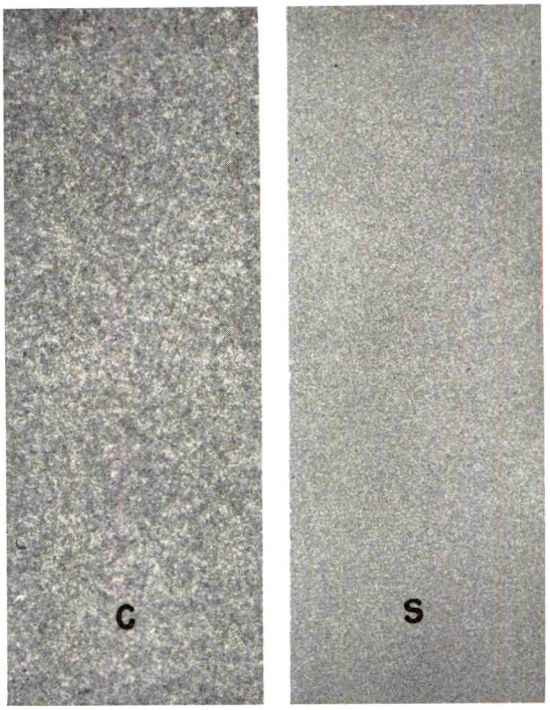


Fig. 1. Intensifying-screen exposure with good contact C, and with screen and film spaced, S. A magnification of 2× and increased contrast were used to make a satisfactory halftone reproduction.

The work of Sturm and Morgan[10] suggests the quantum-mottle explanation. It may be that a point has been reached in the "speed" of film—screen combinations at which so few x-ray photons are *absorbed* per unit area of the screen for an exposure that the natural randomness in their spatial distribution produces the visible screen mottle on the processed film. Roentgen rays are known to consist of discrete entities called "photons," which are "quanta" of electromagnetic energy. (This is also true of ordinary light.) Since these arrive in a random manner, the *average* number absorbed per unit area, *e.g.*, per square millimeter, in a screen will seldom be the same as the *actual* number in a specified square

* A moiré pattern is seen when, for instance, two window screens are superposed slightly out of register. Similarly, any two *identical* patterns so superposed yield a *new gross* pattern. The production of a moiré pattern can thus be used as a sensitive indicator of the similarity of two patterns.

millimeter. For example, if a handful of sand is sprinkled onto a checkerboard, the *average* number of grains per square would be the total number divided by the number of squares, but it would rarely be found that this number actually fell on any one square. It is well known from statistical studies that the *per cent fluctuation* in the *actual* number of grains of sand on a square becomes greater the smaller the *average* number per square. The number of x-ray photons *absorbed* per square millimeter of a screen is analogous to the number of grains of sand per square of the checkerboard. We emphasize that in our experiments, we deal only with the absorbed photons because only those absorbed can cause the screen to fluoresce.

A convenient way of describing the fluctuation in number of absorbed photons is by means of simple mathematical relations derived from statistical theory. If $\bar{n}$ is the average number of photons absorbed per unit area, the "standard deviation" σ is $\sqrt{\bar{n}}$. The relative fluctuation $\sigma/\bar{n}$ is therefore $\sqrt{\bar{n}}/\bar{n}$ or $1/\sqrt{\bar{n}}$. Thus, if the average number of x-ray photons absorbed per square millimeter is 10,000, the relative fluctuation is 1/100 or 1 per cent; but if only 100 are absorbed, it is 1/10 or 10 per cent. In the latter case, statistical theory shows that, on the average, 68 per cent of the unit areas will absorb between (100–10) and (100+10) photons and only 32 per cent will exceed these limits. The quantity $1/\sqrt{\bar{n}}$, therefore, is a measure of the variability of a random occurrence; in this case, the variation to be expected in the number of x-ray photons absorbed per unit area. In terms of visible mottle, the fluctuation would be much more prominent for a small number absorbed than for a large number. We treat the light emission from the screen in terms of the number of *x-ray* photons absorbed because *each* absorbed x-ray photon causes the emission of several hundred blue-light photons.[3]

Realizing that structure mottle might be camouflaged by quantum mottle, an experiment was devised to test this point. The same procedure was used as that which was explained earlier except that Kodak Fine Grain Positive Film was used as a recorder instead of Blue Brand. This film is also sensitive to blue light but is about 1/100 the speed of Blue Brand. Therefore, to produce the same density on this film, a 100 times greater exposure was required, or 100 times as many photons were absorbed per unit area in the screen. Quantum mottle would be much reduced in this slower system but structure mottle would be unaffected. Superposition of the enlargements from the two Positive Films did show a definite, but faint, moiré pattern, thus proving the existence of structure mottle. It should be borne in mind, however, that the use of such a slow film to obtain a faint pattern means that it will be relatively insignificant when using a conventional fast, screen-type x-ray film.

In experimental work it is very desirable to alter but one condition or factor at a time in order to facilitate interpretation of results. We wished to slow down the x-ray film or screens without affecting other properties, such as the sensitivities of the grains themselves. Quantum mottle should be lessened if the screen–film combination is made effectively slower because the number of absorbed x-ray photons required to produce a given density is increased. An experimental intensifying screen of thallium-activated potassium iodide KI(Tl) was chosen for this experiment because the slowing down of such a fast system should produce an effect even greater than that produced by commercial screens.* Screens made of this compound emit about three and one-half times as many light photons per x-ray photon absorbed as did the calcium tungstate screens used. The KI(Tl) screen we used had slightly lower absorption in the medical range of kilovoltages than the calcium tungstate screen because its higher mass absorption was more than offset by its lesser mass per

* We are indebted to Dr. G. B. DeLaMater of the Mallinckrodt Chemical Works for his courtesy in supplying this experimental screen.

unit area. The net effect is that this screen required about one-third the exposure in milliampere-seconds, in the medical range, compared to some of the most widely used calcium tungstate screens. Commercial films and screens are usually given a superficial coating of inert material to protect them against abrasion. By dyeing the protective coating of either the film or the intensifying screen with a dye that absorbs screen light, the number of absorbed x-ray photons necessary to produce a given density is increased without altering any of the basic screen or film characteristics. A yellow dye was successfully applied to the surface of the KI(Tl) screen, thus providing a strong absorber for the screen's light. The most uniformly dyed screen we obtained required an increase in exposure of about 14 times compared with the undyed. The emission pattern of this screen on a single emulsion of Blue Brand Film showed no visible mottle, which lends support to the theory that the mottle from the undyed screen was produced by statistical fluctuation, *i.e.*, it is quantum mottle.

Experimental coatings of a Blue Brand emulsion were made both with and without a yellow dye in the overcoating. The concentration of dye was such that the dyed film had an effective speed of only one-quarter that of the control when exposed to the light of a KI(Tl) experimental screen. The mottle was less evident on the dyed film than on the control. In this case, with the required number of absorbed x-ray photons increased fourfold, there was still some mottle apparent, an amount about the same as in the case of a calcium tungstate screen used with an undyed Blue Brand Film.

The degree of screen mottle produced by a low-intensity exposure (100 seconds to produce $D=0.5$ on Blue Brand) was found to be less than that of a high-intensity exposure (about 1/10 second). It was known in this case that, because of reciprocity failure, the film was about 2.4 times slower at the long exposure time than at the short. Thus, the low-intensity exposure required 2.4 times as many absorbed x-ray photons to produce a given density as did the high-intensity exposure. The change in mottle with exposure time, therefore, is in the proper direction to be explained on a statistical basis since more absorbed photons were necessary to produce the same density in the lower-intensity exposure.

Since the light produced per absorbed x-ray photon in an intensifying screen increases with the quantum energy of the x-ray photon,[5] fewer high-energy than low-energy photons are required to produce a given density. Statistically, if fewer photons are required, the variation in exposure from area to area is greater. We would then expect the mottle to be more prominent for the high-energy than for the low-energy exposure if it is caused by the statistical variation in number of absorbed x-ray photons from area to area.

Exposures were made, using Blue Brand Film and a single calcium tungstate screen, to heavily-filtered x-rays generated at 70 kv. and 180 kv. About 2.7×10^4 photons/mm.^{2} were absorbed in the screen to produce a density of 0.6 with 180 kv. radiation and about 4.7×10^4 photons/mm.^{2} at 70 kv. Thus the high-energy exposure required only about 60 per cent as many photons per unit area to produce a given density as did the low-energy exposure. The mottle was more evident on the high-energy exposure than on the low. This change in the degree of mottle with quantum energy supports the theory that screen mottle is caused largely by the statistical fluctuation in the spatial distribution of the absorbed x-ray photons.

Structure mottle, as opposed to quantum mottle, should be eliminated by introducing irregular relative motion between the film and the screen during the exposure. Since intimate film–screen contact is essential to record the mottle pattern, continuous motion during exposure was not attempted but was approximated by dividing the exposure into a number of equal installments, and moving the film after each installment.

A single emulsion of Blue Brand Film in contact with an experimental KI(Tl) screen was given 10 successive 1/10 second exposures, with the film moved 1/16 to 1/2 inch relative to the screen after each exposure. As a control, a single emulsion of Blue Brand Film was given 10 successive 1/10 second exposures without relative film–screen motion.

The film that was moved between exposures had only slightly less mottle than the control. In fact, the difference was so slight that not all observers agreed that such a difference existed. It thus appeared that the major part of the pattern was from a random cause and not due to a structure of the screen. If this procedure is a valid approximation of continuous motion during exposure, it must be concluded that only a small part of the pattern is inherent in the screen and that most of it results from the random fluctuations.

Most of the experiments which were designed to enable us to find this origin of screen mottle have been of a qualitative nature and therefore no one of them is adequate to furnish "proof." In the aggregate we believe that they do give us proof. At the present time, we can cite one example of a semiquantitative determination of the uniqueness of the statistical nature of most of the screen mottle. (Results of recent quantitative work are to be published.)[8] From microdensitometer traces of a screen-exposed Blue Brand Film it was estimated that the average deviation from the mean (σ) of the uneven density pattern was about ± 0.03 in density. The average net density was 0.30. Since the gradient of the film at this density is about 1.0, the log of the average variation in light intensity was 0.03 or ± 7 per cent.

By assuming that the energy of each absorbed x-ray photon was that of a photon of the "effective wavelength" of the filtered continuous x-ray spectrum and by measuring the absorption of the screen, it was found that approximately 10^4 absorbed x-ray photons per square millimeter are necessary to produce a density of 0.30 on Blue Brand Film.*

The area of the scanning aperture of the microdensitometer was 0.0088 sq. mm., an area which represented the absorption of 88 photons, on the average. The relative fluctuation, $1/\sqrt{88}$, in this case, is 11 per cent. We consider this good agreement with the calculated value of 7 per cent as the experimental approximations which were made would lead us to expect a fairly large error.

So far, results of our experiments point unmistakably toward the strong preponderance of quantum mottle over structure mottle in the final screen mottle disclosed by the recording film. The question may well be raised whether there are any experiments with such results as to place some doubt on this conclusion. We have found that a "fast screen," that is, one in which the x-ray exposure is relatively low to obtain a given film density, does not necessarily yield a bad mottle. Suppose that the speed of the screen is achieved by using a thick phosphor layer, *e.g.*, calcium tungstate, to obtain high x-ray absorption. On a statistical basis, this should not alter the mottle pattern produced by a given phosphor material, since no matter what the x-ray absorption or the screen thickness[7] (neglecting the light absorption and scatter within the screen) the same *number* of scintillations is required to produce a given density. The thick phosphor layer, however, allows the scintillations to diffuse or spread before they are recorded on the film and thus reduces the degree of mottle. For this same reason, the "sharpness" of the screen is adversely affected so that both the image of the anatomic part and the screen mottle are less sharp.

The converse of the thick-screen argument probably applies to slow screens which show detail well; that is, the calcium tungstate phosphor layer is so thin that the individual scintillations are recorded with very little image spread. Such a reduction in image spread represents improved sharp-

* This is in good agreement with the findings of Sturm and Morgan.[10] They calculated that Blue Brand Film exposed between Patterson Par Speed intensifying screens required 4×10^4 photons per square millimeter to produce a density of 1.0.

ness of a screen. Since, from the film's "point of view," quantum mottle is an inseparable part of the image, an increase in the degree of mottle must accompany an improvement in the definition of the screen. This implies that, for a given screen phosphor, the degree of mottle might be used as a measure of the definition of the screen provided a material has not been added that changes the apparent efficiency of the phosphor. We use the word "efficiency" here to mean the amount of light generated per absorbed x-ray photon.

Our tests have shown that the efficiency of the experimental KI(Tl) screens which we used was greater than that of the calcium tungstate; that is, for the same x-ray absorption in the screen there was a greater light output from these screens. Thus, each x-ray photon which was absorbed in the potassium iodide screen yielded more light than a photon absorbed in calcium tungstate. Since fewer photons are absorbed for the same film density, their statistical fluctuation is greater, and the greater screen mottle of the iodide screen is thus explained.

So far, we have looked upon the film simply as a recorder of the screen pattern yielding density fluctuations which we see and call "screen mottle." A factor in determining the visibility of screen mottle is the contrast of the film. As is well known, image structures stand out more prominently in a radiograph made on a film of high contrast than they do on one made with a film of low contrast. Similarly, the prominence of screen mottle is dependent on film contrast. Whether this has a significant bearing on the diagnostic value of a radiograph, we do not know. In the language of the communications engineer, we should only expect improvement if the signal-to-noise ratio is increased. The most obvious way to improve signal-to-noise ratio is to use a slow film, other factors remaining the same. The slow film will require the absorption of a relatively large number of x-ray photons in the screen, thus yielding a low quantum mottle. Of course, the requirements in a given radiographic technique may dictate the use of a fast film and the greater amount of quantum mottle must then be accepted.

It is generally known that in *direct* x-ray exposures, the graininess of a film increases with increasing kilovoltage applied to the x-ray tube and with increasing filtration of the beam. The theory of this increase of graininess with increasing quantum energy of the exposing radiation, as first given by Eggert and Schopper,[4] was based on the increasing range of the photoelectric and Compton recoil electrons in the emulsion. Their theory was supported by Bromley and Herz,[1] who showed that the number of silver bromide grains made developable per absorbed x-ray photon increased with quantum energy. Further support was given by Seemann's[9] work, in which the effect measured was the photographic density. Thus, the photographic effect on the emulsion, *per quantum absorbed*, increases with increasing energy of radiation, and direct x-ray graininess therefore may also be viewed qualitatively as a *statistical phenomenon*. This does not, however, invalidate Eggert and Schopper's explanation of the *physical* process involved.

CONCLUSIONS

The screen mottle observed on high-speed x-ray films exposed with fluorescent intensifying screens arises primarily from statistical fluctuations in the spatial distribution of the absorbed x-ray photons (quantum mottle) and secondarily from inhomogeneities in the phosphor coating (structure mottle). On this basis, the major contributing factors that control the variations in the degree of mottle from screen to screen are (1) the inherent efficiency of the phosphor material; (2) the optical properties of the phosphor layer, such as the transparency of the crystals or the presence of materials, such as dyes, that alter their effective efficiencies by absorbing some of the light; and (3) the thickness of the phosphor coating. The less efficient phosphors, or those with lower effective efficiency, produce a lower degree of mottle for a given x-ray absorption in the screen. Among

screens differing only in thickness, the thicker ones give the lesser mottle, even with their higher speed, because of their greater diffusion of light.

Characteristics of x-ray film which are factors in determining the prominence of screen mottle are (1) the speed of the film, *i.e.*, a quantity which is inversely proportional to the amount of screen light (or number of absorbed x-ray photons) required to yield a specified density, and (2) the contrast of the film; the greater the contrast the more readily visible are the useful parts of the image as well as screen mottle.

The increase of graininess of direct x-ray exposures as the quantum energy of the exposing radiation increases can be attributed qualitatively to statistical fluctuations in the spatial distribution of absorbed x-ray photons.

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THE FREQUENCY RESPONSE FUNCTION*

A VALUABLE MEANS OF EXPRESSING THE INFORMATIONAL RECORDING CAPABILITY OF DIAGNOSTIC X-RAY SYSTEMS

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THERE can be little question that the value of a radiologic technique is largely determined by the quantity of diagnostic information it yields. So important is this, that physicians who use radiologic methods in their practice must be fully aware of the factors which govern the informational recording capability of x-ray systems. This is particularly so if one wishes to achieve maximal information with minimal patient exposure.

In general, the diagnostic information recorded by a film or screen is governed by (a) the characteristics of the anatomic structure under examination, (b) the ability of the radiographic or fluoroscopic system to record the information impressed upon it and (c) a number of physiologic and psychologic parameters associated with the observer. It is the purpose of this paper to examine the second of this series of factors, namely, the informational recording capability of x-ray systems.

Qualitatively, it is well known that the diagnostic information contained in a film or screen is a function of the ability of the film or screen to record fine detail; that is, the ability of the recording material to reproduce the images of anatomic structures of small size with maximal sharpness and minimal blurring. Extensive reserach has shown that the ultimate limit of a film or screen to record fine detail is governed by statistical fluctuations in the number of x-ray photons impinging on and absorbed by the film or screen during exposure. Quantitatively, this relationship may be expressed by the equation,

$$\delta = \frac{k}{C(nt)^{1/2}} \qquad (1)$$

where δ is the diameter in cm. of the smallest perceptible image, k is a constant having a value of about 200, C is the per cent contrast of the image, n is the number of x-ray photons per sq. cm. absorbed each second by the intensifying or fluoroscopic screen and t is the exposure time in seconds in radiography and the storage time of the eye (about 0.2 sec.) or of the system, whichever is greater, in fluoroscopy. As stated, equation (1) indicates the ultimate limit of an x-ray system to record fine detail; that is, the limit beyond which one cannot improve system performance regardless of what one does.

The limits imposed by photon statistics on radiologically derived information are seldom reached in practice. This is due to certain deficiencies in the characteristics of radiographic and fluoroscopic systems and to less than ideal conditions under which these systems are usually viewed. Hence, the radiologic process generally yields less diagnostic information than is theoretically possible. A corollary of this, implied by equation (1), is that more x-ray photons and therefore higher patient doses are required to reach a particular level of diagnostic information than would be the case if these defects were not present.

The inability of radiographic and fluoroscopic systems to achieve the limits of performance set by statistical theory may be traced to a number of factors. First, the efficiency with which most fluorescent

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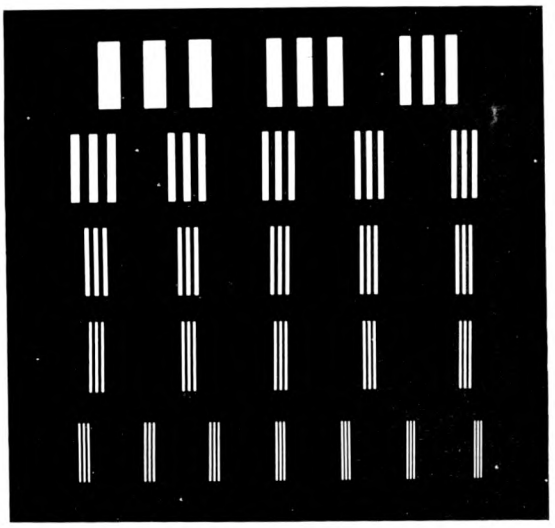


FIG. 1. Line test pattern used in the measurement of photographic film resolution.

materials convert x-rays to light is rather low. As a result, the crystals of which these materials are composed must be relatively large to achieve a satisfactory light output. In addition, fluorescent screens must have a thickness of several crystal diameters for satisfactory absorption of the incident radiation. Hence, considerable diffusion of light occurs beyond the region where each x-ray photon is absorbed. Because of these two factors, the resolution of the x-ray process is impaired and sometimes substantially so. This pertains to every application of fluorescent materials in radiology: in radiography, conventional fluoroscopy, fluoroscopy aided by image amplifiers, photofluorography and cinefluorography.

RESOLUTION AND UNSHARPNESS

In photographic physics, the ability of a film to record detail is customarily measured by focusing on it a test pattern, consisting of a graded series of lines and spaces, equal in width, similar to that shown in Figure 1. Resolution or ability to record detail is then expressed as the maximum number of lines per millimeter which one may observe in the film. In radiology, Van Allen and Morgan[4] have devised an instrument by which a similar pattern may be impressed on a radiographic film for the measurement of its resolution. The apparatus, shown schematically in Figure 2, consists of a slit system formed by lead-alloy blocks mounted on a carriage which can be moved laterally by a differential screw. The width of the slit may be adjusted by a second differential screw. The mechanism rests on the cassette which includes the film or film-screen combination under test. The slit system is first adjusted to a width corresponding to slightly fewer lines per millimeter than the expected resolving power of the film or film-screen combination, then an x-ray exposure is made. Next, the slit system is moved twice the width of the slit and another exposure is made and

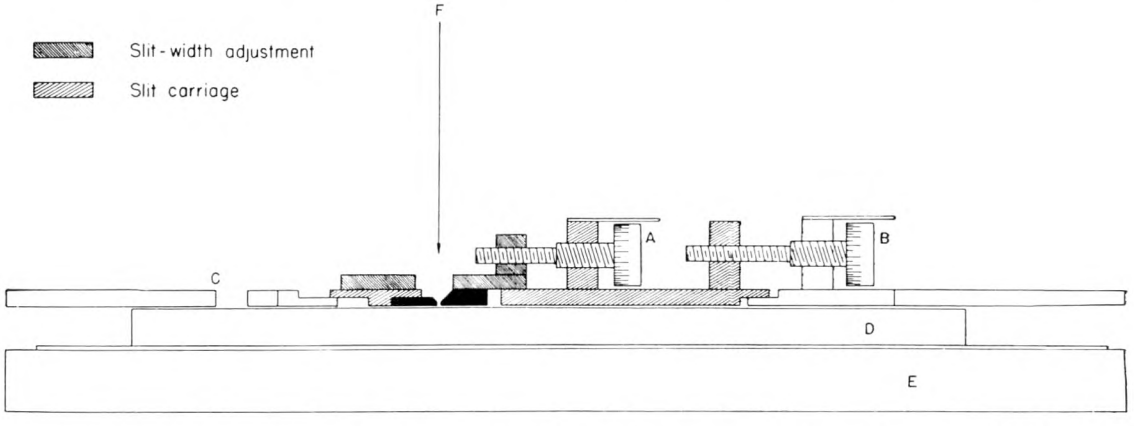


FIG. 2. Schematic diagram of mechanism to produce line test pattern on radiographic materials (after Van Allen and Morgan[4]). A, slit adjustment screw; B, slit position screw; C, test exposure port; D, cassette; E, instrument platform; F, x-ray beam.

so on until a group of 4 or 5 lines is produced. That is, a line-space pattern of the same type as that used in photographic practice is produced on the film. The slit system is then adjusted to correspond to a higher resolving power by narrowing the slit, and another series of exposures is made as before. After this procedure has been repeated several times for progressively finer patterns, the film is processed and examined under low-power magnification. The resolution is taken as the number of lines per millimeter in that group of exposures in which the lines are just distinguishable.

The size of the crystalline structure of fluorescent materials is not the only factor which restricts the resolution of the radiologic process. Among others, may be included the finite size of the focal spot of the x-ray tube and movement of the anatomic structure under examination.

The manner in which the focal spot of an x-ray tube causes blurring or unsharpness is shown in Figure 3 where O is an object under examination, F is the focal spot and R is a radiographic film or fluoroscopic screen. It is apparent from the diagram that the image boundary of O, appearing on the film, is spread over the region from x_0 to x_1, and hence is blurred. The extent of this blurring is expressed quantitatively by the equation

$$U_g = x_1 - x_0 = \frac{Fd_2}{d_1} \qquad (2)$$

where F is the cross-section of the focal spot, d_1 is the distance from focal spot to object and d_2 is the object-film distance.

The effect of anatomic movement on image clarity is illustrated in Figure 4 where O is an object which moves from A to B during exposure, F is the focal spot of the x-ray tube and R is the radiographic film. It is evident that the motion of O causes its image boundary to be blurred over the region of the film from x_0 to x_1. The amount of this blurring or unsharpness is given by the equation

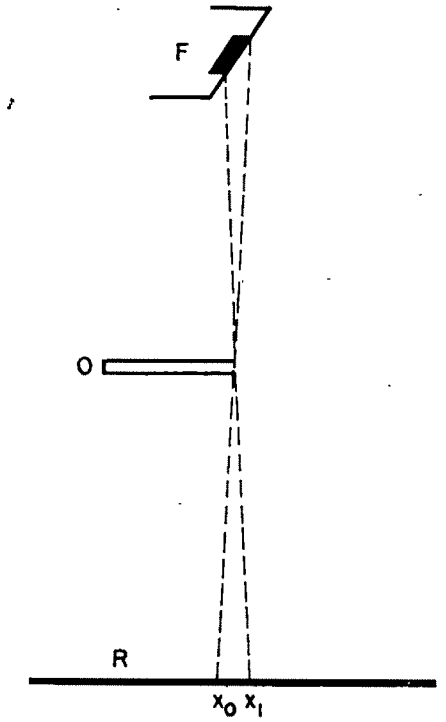


FIG. 3. Schematic diagram illustrating manner in which focal spot of x-ray tube causes blurring of x-ray images.

$$U_m = x_1 - x_0 = \frac{vt(d_1 + d_2)}{d_1} \qquad (3)$$

where v is the velocity of the object, t is the exposure time and d_1 and d_2 are as before.

The methods, described in foregoing paragraphs for the measurement of (a) the resolution of fluorescent materials and (b) the unsharpness created by the x-ray focal spot and anatomic movement, have only limited value in the determination of the informational recording capability of an x-ray system. Although it is true that films and screens which exhibit high resolution and small amounts of unsharpness generally yield more information than those which do not, a quantitative evaluation of the over-all capability of an x-ray system to record detail cannot be made from such measurements. This is due in part to their failure to provide data which fully characterize the recording performances of the various components of an x-ray system and in part to the fact that unsharpness and

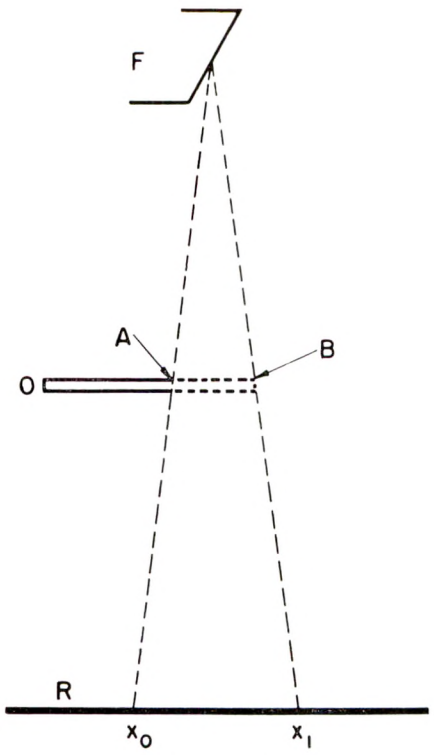


Fig. 4. Schematic diagram illustrating manner in which anatomic motion causes blurring of radiographic images.

resolution values are expressed in terms which are dimensionally different from one another.

To circumvent these difficulties, a number of investigators have recently turned to the frequency response function as a method of expressing the recording capability of x-ray system components. Frequency response data have now been published on a few types of fluoroscopic screens, x-ray films and image amplifiers.[3] In subsequent paragraphs, the frequency response functions generated by x-ray focal spots and anatomic motion will be developed.

THE FREQUENCY RESPONSE FUNCTION

The use of the frequency response function is relatively new in radiology and hence a discussion of its derivation is perhaps in order. For this, let us again consider the test pattern illustrated in Figure 1. As we have seen, it consists of several groups of lines and spaces which, when recorded on film, produce images whose densities rise and fall across the film through maxima and minima corresponding to the lines and spaces of the pattern. Hence, the densities exhibit a periodicity or *spacial frequency* which may be expressed quantitatively in terms of the number of cycles or lines recorded per unit length of the film.

When the test pattern is coarse, the film is usually able to reproduce it with good clarity and relatively high contrast. When the line structure becomes finer, however, image clarity deteriorates and measurement of the densities of the film indicates that the contrast between lines and spaces has decreased; that is, the film exhibits a diminished response to the variations in light intensity impressed upon it by the test pattern. As the pattern becomes still finer, there ultimately comes a time when line structure is no longer visible in the film. The line-space contrast disappears and film response to the test pattern has clearly fallen to zero. Now the ratio of the line-space contrast prevailing at a given frequency of the image pattern to the maximum contrast observed at any frequency is a measure of the relative response of the film. When such ratios are plotted against appropriate frequency values, there results a relationship which expresses film response as a function of the spacial frequency of the image pattern. *Such a relationship is called a frequency response function.*

Figure 5 illustrates the frequency response function of a typical fluoroscopic screen. It indicates that this screen records well the coarse patterns of low spacial frequency. As frequency increases (*i.e.*, the line pattern becomes finer), the screen's contrast response falls and recording capability deteriorates. It will be observed that at a frequency of approximately 6 cycles per mm., the response of the screen drops to 1 per cent. This is interesting because the threshhold contrast perception of the human eye[2] is near 1 per cent; furthermore,

measurements of the resolution of fluoroscopic screens made by Van Allen and Morgan[4] have shown these screens to have a resolving power of about 5 lines per mm. Hence, it appears that conventional resolution measurements simply constitute but one point on the frequency response curve of a recording material.

Ideally, the frequency response function of a film or screen should be measured with a test pattern which produces an image whose intensity varies sinusoidally from point to point. Such a test pattern is extremely difficult to construct and hence measurements have been conducted with square wave patterns of sharp lines and spaces. The difference in the two response functions is not great, however.

A. FREQUENCY RESPONSE FUNCTION OF X-RAY TUBE FOCAL SPOT

The manner in which the frequency response function of an x-ray tube focal spot is generated is shown schematically in Figure 6 where O is a test object constructed in such a manner that its x-ray transmission varies sinusoidally from point to point in accordance with the relationship

$$T = T_0(1 - \sin 2\pi f_0 x) + T_1 \qquad (4)$$

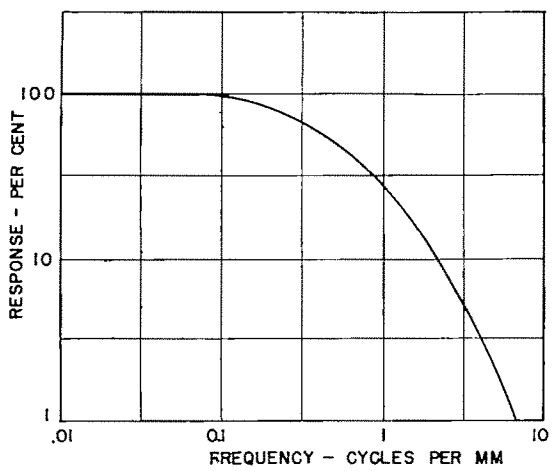


Fig. 5. Frequency response function of a typical fluoroscopic screen (after Oosterkamp and Albrecht[3]).

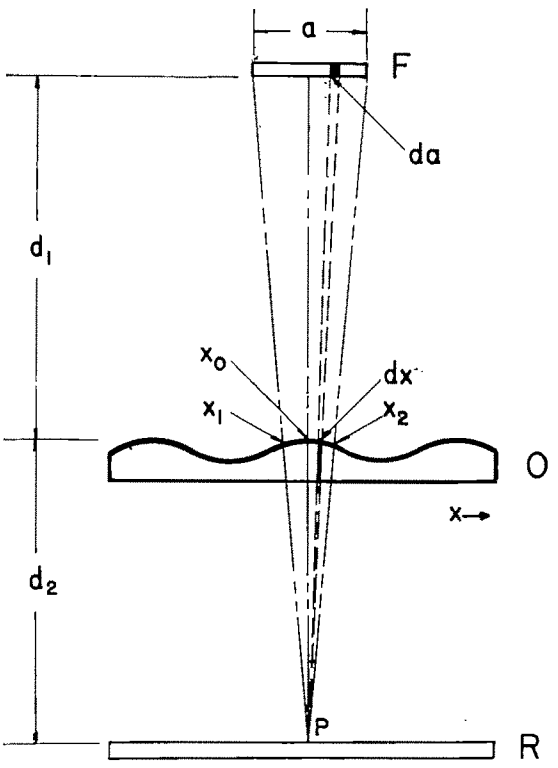


Fig. 6. Schematic diagram illustrating manner in which frequency response function of x-ray focal spot is generated.

where T is x-ray transmission, f_0 is the spacial frequency of the test object in cycles per millimeter, x is the position in the test object where T is measured and T_0 and T_1 are constants such that $2T_0+T_1 \leq 1$. The test object is irradiated by the x-ray tube (F) of focal-spot cross-section (a) and recorded by the radiographic film (R). The object-focal spot distance is d_1 and the object-film distance is d_2.

It will be observed that the incremental radiant intensity, dG, received at point P of the film from the focal spot element, da, is given by the equation

$$dG = G_a T \cdot da \qquad (5)$$

where G_a is the radiant intensity which would be delivered to point P from unit length of the tube's focal spot if no test pattern were present.

It will be observed from simple geometric principles that

$$da = \frac{d_1 + d_2}{d_2} \cdot dx. \tag{6}$$

Therefore, when equations (4) and (6) are substituted in equation (5),

$$dG = G_a \left[\frac{d_1 + d_2}{d_2}\right] \cdot [T_0(1 - \sin 2\pi f_0 x) + T_1] \cdot dx. \tag{7}$$

The radiant intensity, G, received from the entire focal spot of the x-ray tube may be obtained by integration of equation (7) over the interval of x from x_1 to x_2. That is,

$$\begin{aligned} G &= \int_{x_1}^{x_2} G_a \left[\frac{d_1 + d_2}{d_2}\right] \cdot [T_0(1 - \sin 2\pi f_0 x) + T_1] \cdot dx \\ &= \left[\frac{G_a T_0}{2\pi f_0}\right]\left[\frac{d_1 + d_2}{d_2}\right] \cdot (\cos 2\pi f_0 x_1 - \cos 2\pi f_0 x_2) \\ &\quad + G_a \left[\frac{d_1 + d_2}{d_2}\right](T_0 + T_1)(x_2 - x_1). \end{aligned} \tag{8}$$

Now an examination of Figure 6 reveals that

$$(d_1 + d_2)/d_2 = a/(x_2 - x_1) \tag{9}$$

and that

$$x_1 + x_2 = 2x_0 \tag{10}$$

where x_0 is the point in the test object through which a line joining point P of the film and the center point of the focal spot passes. Furthermore, it may be shown by classic trigonometric relationships that

$$\cos 2\pi f_0 x_1 + \cos 2\pi f_0 x_2 = 2 \sin \pi f_0(x_1 + x_2) \cdot \sin \pi f_0(x_2 - x_1). \tag{11}$$

Therefore, equation (8) becomes

$$G = \left[\frac{G_a T_0 a}{\pi f_0(x_2 - x_1)}\right] \sin 2\pi f_0 x_0 \cdot \sin \pi f_0(x_2 - x_1) + G_a(T_0 + T_1)a. \tag{12}$$

Equation (12) indicates that the image of the test object illustrated in Figure 6 exhibits a pattern in which the radiation intensity varies sinusiodally from point to point across the film from a maximum value of

$$G_a(T_0 + T_1)a + \left[\frac{G_a T_0 a}{\pi f_0(x_2 - x_1)}\right] \cdot \sin \pi f_0(x_2 - x_1)$$

to a minimum value of

$$G_a(T_0 + T_1)a - \left[\frac{G_a T_0 a}{\pi f_0(x_2 - x_1)}\right] \cdot \sin \pi f_0(x_2 - x_1).$$

The contrast C between the greatest and least intense components of the image is therefore,

$$C = \frac{2G_a T_0 a \cdot \sin \pi f_0(x_2 - x_1)}{\pi f_0(x_2 - x_1)} \cdot \tag{13}$$

An inspection of equation (13) indicates that contrast is maximal when the spacial frequency f_0 of the test object approaches zero; that is when the test pattern is coarsest. Under these circumstances

$$C_m = 2G_a T_0 a \tag{13a}$$

where C_m is maximal contrast.

Hence, when contrast is expressed as a fraction of maximal contrast, equation (13) becomes

$$C_f = \frac{\sin \pi f_0(x_2 - x_1)}{\pi f_0(x_2 - x_1)} \tag{14}$$

and when system parameter values, given in equation (9), are substituted for the term, $x_2 - x_1$,

$$C_f = \frac{\sin \pi f_0 a d_2/(d_1 + d_2)}{\pi f_0 a d_2/(d_1 + d_2)} \cdot \tag{15}$$

Equation (15) is the frequency response function of a radiologic system due to the focal spot of the x-ray tube when expressed in terms of the spacial frequency of the object under study. As indicated, system response is maximal at low frequencies. As the spacial frequency increases, however, the response diminishes until, at a frequency equal to $(d_1+d_2)/d_2 a$, system response falls to zero and the x-ray image disappears. At still higher frequencies, the response rises and falls through a series of

cyclical excursions, each one of which exhibits a maximal response less than its predecessor. These realtionships are shown graphically in Figure 7.

Sometimes it is desirable to express the frequency response function of the x-ray focal spot in terms of the spacial frequency of the image as it appears on the film or screen rather than in terms of the frequency of the object under study. For the system illustrated in Figure 6, the relationship between the two frequencies may be expressed by the equation

$$f_0/f_i = (d_1 + d_2)/d_1 \qquad (16)$$

where f_i is the spacial frequency of the image. Therefore from equation (15), the required frequency response function due to focal spot size when expressed in terms of image frequency, is

$$C_f = \frac{\sin \pi f_i a d_2/d_1}{\pi f_i a d_2/d_1} . \qquad (17)$$

The lowest image frequency at which system response falls to zero is d_1/d_2a. For a focal spot of 2 mm. cross-section, an object-film distance of 10 cm. and an object-focal spot distance of 90 cm., this frequency is 4.5 lines or cycles per mm.

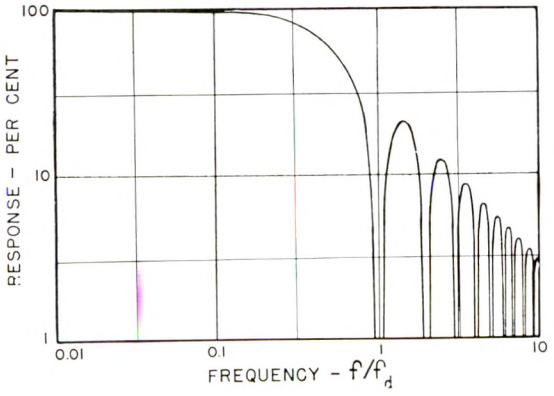


Fig. 7. Frequency response functions of x-ray focal spot and anatomic motion. For x-ray focal spot functions, f_d equals an object frequency of $(d_1+d_2)/d_2a$ or an image frequency of d_1/d_2a cycles per mm. For anatomic motion functions, f_d equals an object frequency of $1/vt$ or an image frequency of $d_1/(d_1+d_2)vt$ cycles per mm.

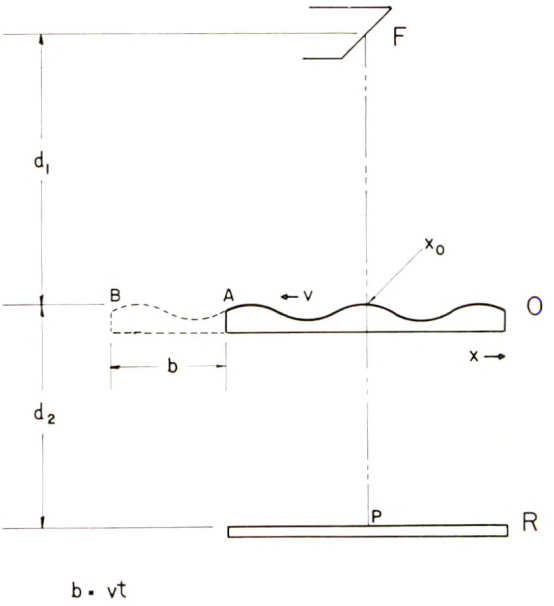


Fig. 8. Schematic diagram illustrating manner in which frequency response function of anatomic motion is generated.

B. Frequency Response Function due to Motion

The influence of anatomic motion on the frequency response of a radiologic system is illustrated schematically in Figure 8. As in Figure 6, O is a test object, constructed in such a manner that its x-ray transmission varies sinusoidally from point to point in accordance with equation (4). Exposure is made with an x-ray tube (F) and the image of the object is recorded by the radiographic film (R). As before, d_1 is the distance between object and x-ray tube focal spot and d_2 is the distance between object and film. At the beginning of the exposure, point x_0 of the test object is directly over point P of the film. During the time of exposure t the test object moves with a uniform velocity v from position A to position B, a distance of b mm. This distance is equal to the product vt.

The incremental exposure dE received at point P of the film during an exposure time dt is given by the familiar relationship

$$dE = G \cdot dt \qquad (18)$$

where G is the intensity of the radiation

falling on the film at the time of exposure. From Figure 8, it is clear that this intensity is given by the expression

$$G = G_x T \tag{19}$$

where G_x is the intensity which would fall on the film if no test object were present. When equations (4) and (19) are substituted in equation (18),

$$dE = G_x T_0(1 - \sin 2\pi f_0 x)\cdot dt + G_x T_1 \cdot dt. \tag{20}$$

Now the velocity v of a uniformly moving object is equal to dx/dt. Therefore equation (20) becomes

$$dE = \left[\frac{G_x T_0}{v}\right](1 - \sin 2\pi f_0 x)\cdot dx + \left[\frac{G_x T_1}{v}\right]\cdot dx. \tag{21}$$

When equation (21) is integrated over the interval between x_o and x_0+vt, the total exposure received by point P is given by the relationship

$$E = \int_{x_o}^{x_0+vt}\left[\frac{G_x T_0}{v}\cdot(1 - \sin 2\pi f_0 x) + \frac{G_x T_1}{v}\right]\cdot dx$$
$$= \left[\frac{G_x T_0}{2\pi f_0 v}\right][\cos 2\pi f_0 x_0 - \cos 2\pi f_0(x_0 + vt)]$$
$$+ G_x(T_1 + T_0)t. \tag{22}$$

Through the use of the same trigonometric relationships from which equation (11) was derived, equation (22) may be written

$$E = \left[\frac{G_x T_0 t}{\pi f_0(vt)}\right]\sin \pi f_0(2x_0 + vt)\cdot \sin \pi f_0(vt)$$
$$+ G_x(T_1 + T_0)t. \tag{23}$$

It will be observed that equations (23) and (12) are of similar form. Hence, the frequency response function of a radiologic system due to anatomic motion, expressed in terms of the spacial frequency of the test object, is

$$C_f = \frac{\sin \pi f_0 vt}{\pi f_0 vt}. \tag{24}$$

Through equation (16), the response expressed in terms of the image frequency at the plane of the film is

$$C_f = \frac{\sin \pi f_i vt(d_1 + d_2)/d_1}{\pi f_i vt(d_1 + d_2)/d_1}. \tag{25}$$

The frequency response functions due to anatomic motion are similar in form to those generated by the focal spot of an x-ray tube. Hence, the relationships expressed in Figure 7 are equally valid for anatomic motion. As the spacial frequency increases, the response diminishes until, at an object frequency of $1/vt$ or an image frequency of $d_1/(d_1+d_2)vt$, system response falls to zero and the x-ray image disappears. For an object moving at a rate of 5 mm. per sec., an exposure time of 0.1 sec., an object-focal spot distance of 90 mm. and an object-film distance of 10 cm., the lowest image frequency at which the image disappears is 1.8 lines per mm.

C. ADVANTAGES OF THE FREQUENCY RESPONSE FUNCTION IN EXPRESSING THE INFORMATIONAL RECORDING CAPABILITY OF X-RAY SYSTEM COMPONENTS

Each component of an x-ray system which affects the system's ability to record detail generates a frequency response function. In foregoing paragraphs we have spoken of the functions generated by fluoroscopic screens, the focal spots of x-ray tubes and the motion of anatomic structures. Similar functions may be derived either by measurement or mathematical development for all components, including radiographic, photofluorographic and cinefluorographic film, intensifying and photofluorographic screens, photographic lenses, image amplifiers and television systems. It therefore may be worthwhile at this point to outline the advantages exhibited by frequency distribution functions over resolution and unsharpness measurements in the assessment of an x-ray system's ability to record detail.

It has been previously pointed out that an x-ray material's resolving power constitutes but a single point on the material's frequency response curve. Furthermore,

the measurement is made subjectively by one's observing the finest line-and-space pattern recorded by the material. The frequency response function, on the other hand, is determined instrumentally and, when completed, provides an objective measure of the material's response at many line-space frequencies, thus giving a much broader indication of the material's recording capability. This latter point is of some importance since two components of an x-ray system may exhibit quite different frequency response functions although at one frequency their responses may be identical. If resolution measurements were made at this frequency, they would erroneously indicate the two components to have identical effects on the imaging quality of the system.

The most important advantage of the frequency response function may be found in the fact that a composite frequency response curve, quantitatively depicting the informational recording capability of an entire x-ray system, may be easily derived from the frequency response curves of the several components of the system. This is illustrated graphically in Figure 9, where curve A is the frequency response function of a typical radiographic film exposed with intensifying screens; curve B is the function generated by a 1 mm. focal spot with an object-focal spot distance of 90 mm. and an object-film distance of 10 cm.; and curve C is the function generated by an anatomic structure moving with a velocity of 1 mm./sec. during an exposure time of 0.3 second. Curve D is the system's composite frequency response function. It is derived simply by one's multiplying together the response values indicated by the three curves, A, B and C, at each of several points across the frequency spectrum. This yields a series of composite response values which when plotted against appropriate frequency levels gives rise to curve D.

It will be observed that the composite curve begins to fall at a lower spacial frequency than the individual curves. This, of course, is what one might expect, because

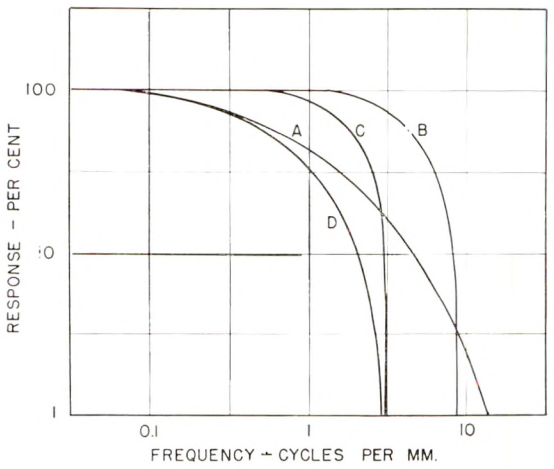


Fig. 9. Individual frequency response functions of the several components of a radiographic system (A, B and C) and the composite frequency response function of all three components operating collectively (D).

each component of a system degrades the image to some extent and the total effect of several components should be greater than that of any one.

Through the use of frequency response functions, it is interesting to observe the influence of various x-ray system components on the ability of a system to record fine detail. As an example, let us consider the case where radiography of the chest is to be performed for evaluation of the vascular structures of a patient's lower lung fields. Let us assume that these structures lie 10 cm. from the film and move during cardiac systole at a velocity of 15 mm. per second. Let us further assume that the x-ray focal spot has a cross-section of 2 mm., is located 180 cm. from the structures under examination and that high milliamperage equipment is available which permits an exposure time of 1/60 second.

The frequency response functions generated by the x-ray focal spot and anatomic motion are shown by curves B and C respectively in Figure 10. Curve A illustrates the function of a typical film-screen combination which might be used in the study and curve D illustrates the composite function of the system. The composite curve indicates that the ability of the system to

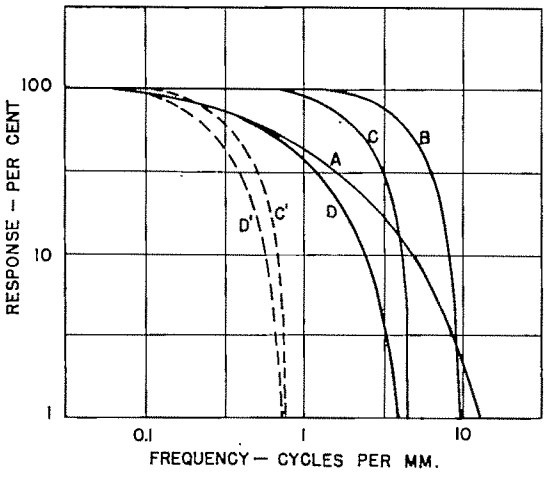


FIG. 10. Effect of varying levels of anatomic motion on the recording capability of a radiographic system.

record vascular structures of 1 mm. or a little less in diameter is relatively good but that anatomic motion prevents visualization of very fine detail.

Let us now consider the results if high-milliamperage equipment were not available and the examination had to be carried out with an exposure time of 0.1 second. The frequency response function due to anatomic motion moves from its original position at C to a new position, C'. This causes the composite function to move to the position D'.

An examination of curve D' reveals that it is not dissimilar in form and position to curve C'. This is characteristic of situations where a number of components of a system limit image quality but where one of them exerts a substantially greater effect than the others; under this circumstance, the composite function approaches the function with the poorest frequency response. It is also evident that the high frequency response of the system has deteriorated substantially from D to D' as the exposure time has increased. Vascular structures must now be several millimeters in diameter to be seen and fine detail is completely missing.

From the foregoing example, one may ask if radiographic films generally should be taken only with high milliamperage equipment. Certainly this is so if one is interested in recording as much diagnostic information as possible. Indeed, an examination of the frequency response function of the film-screen combination shown in Figure 10 reveals that any anatomic motion greater than 0.1 mm. in amplitude during exposure degrades image quality. Such a stringent requirement on anatomic motion may come as a surprise to some since there are many examples where physiologic activity often prevents one from meeting this criterion with any type of x-ray apparatus. This suggests that conventional radiologic practice does not uniformly require a recording capability as great as that currently provided by radiographic films and screens. In fact, even under the conditions of the short exposure time represented by curve C of Figure 10, the film-screen combination's high frequency response is greater than that of the anatomic structure and hence is better than it need be. This in turn suggests that one may often effect important reductions in patient exposure without loss of significant diagnostic information through the use of screens and films of greater sensitivity and of more limited frequency response than those now used.

It is perhaps noteworthy that the value of the frequency response function is by no means limited to conventional radiography. It is equally useful in fluoroscopy, with or without screen intensification, cinefluorography and photofluorography. When such functions have been determined for image amplifiers, lenses, film and television systems, an objective comparison of available cinefluorographic systems will be possible for the first time.

D. FREQUENCY RESPONSE FUNCTIONS IN SYSTEMS WITH MAGNIFICATION OR MINIFICATION

In many radiologic systems, including photofluorography, cinefluorography and televised fluoroscopy, the images to be recorded are magnified or minified during one or more stages of the recording process.

Such changes in image size require special attention when the frequency response functions of the several components of the system are compared.

Whenever a system component introduces a change in image size, the frequency response function of that component and those of all following components must be corrected by a factor equal to this change. For example, in photofluorography, if a fluoroscopic image is recorded by an appropriate lens on photofluorographic film with a six-fold diminution in the size of the original image, the frequency response functions of the lens and of the film must have their frequency scales multiplied by a factor of one-sixth before these functions may be compared with that of the screen. This step is necessary because each frequency contained in the fluoroscopic image is multiplied six-fold by the minification process and hence, correction of the lens and film functions by a factor of one-sixth is required to bring the frequency scales of these functions to a frame of reference common to that of the screen's function.

This procedure is illustrated in Figure 11 where A represents the frequency response function of a fluoroscopic screen, B the uncorrected function of a typical photofluorographic lens and C the uncorrected function of a photofluorographic film. B′ and C′ are the corrected functions of lens and film respectively, obtained by transposing curves B and C to the left until the frequency scale of each function is one-sixth its original value. Curve D is a composite frequency response function of the complete photofluorographic system derived from curves A, B′ and C′.

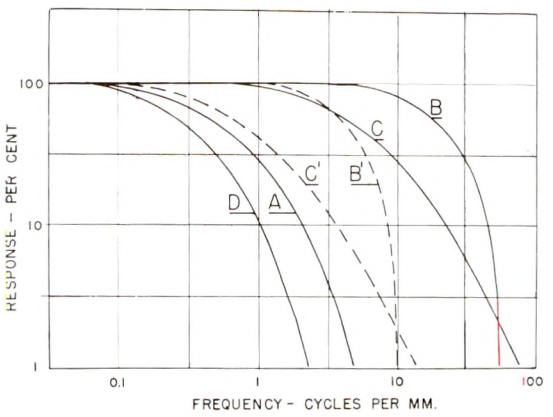


Fig. 11. Illustration of the correction of frequency response functions imposed by image minification in photofluorography.

E. The Relationship of Time-Based and Space-Based Frequency Response Functions

Until now, attention has been devoted entirely to frequency response functions in which frequency has the dimensions of the reciprocal of length (*e.g.*, cycles per mm.). When television techniques are applied to radiologic systems, frequency response functions are encountered in which frequency has the dimensions of the reciprocal of time (*e.g.*, cycles per sec.). The functions of the video amplifiers of television equipment constitute an example of this.

When an evaluation of a radiologic system which includes television apparatus is to be made, it is essential that the time-based functions be converted to space-based functions; that is, all functions must be brought to a common frame of reference. Otherwise, a comparison of the recording capabilities of the various components of the system cannot be carried out and the operational methods to determine the recording capability of the entire system cannot be performed on the several functions.

The relationship between spacial frequency and temporal frequency is expressed by the equation

$$f_s = f_t / f_h s \tag{26}$$

where f_s is spacial frequency, f_t is temporal frequency, f_h is the horizontal scanning frequency of the television system and s is the length of the scanning line.

By means of equation (26), time-based frequency response functions may be easily converted to space-based functions. For example, a video amplifier having a temporal frequency response function which extends to an upper limit of 10 megacycles/sec., operating in a television system with a conventional 15.75 kilocycle/sec. horizontal

scanning frequency and delivering its signals to a kinescope in which the length of the scanning line (width of picture) is nominally 200 mm. has, from equation (26), an equivalent spacial frequency response which extends to an upper limit of approximately 3 cycles per mm. It will of course be remembered that if the width of the kinescopic image is greater or less than the width of the flouroscopic image appearing at the input screen of the x-ray system, this spacial frequency value will require further correction in accordance with the principles set forth in Section D, above.

DISCUSSION AND SUMMARY

The frequency response function is a powerful method by which the informational recording capability of x-ray materials and apparatus may be expressed. By relatively simple techniques, the recording capability of even extremely complex x-ray systems may be easily determined from the functions of individual system components. It is perhaps interesting that although the concept of the frequency response function has been introduced to the field of radiology only recently, such functions have long been used as a means to evaluate the performance of equipment in other physical sciences, particularly in the field of electronics.

In this paper, a few examples of the application of the frequency response function to radiologic problems have been given. Full use of this technique, however, must await the publication of frequency response data on the broad range of materials and apparatus available to the profession. To obtain such data, a diagnostic standards laboratory has been established in the Department of Radiological Science of The Johns Hopkins University with the support of the Division of Radiological Health, Public Health Service. It is hoped that when full operation of the laboratory is reached, the radiologist will be provided with data of a sufficiently comprehensive nature that radiographic and fluoroscopic techniques may be easily chosen which will yield maximal diagnostic information with minimal patient dose.

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Sixty-third Annual Meeting: Shoreham Hotel, Washington, D. C., October 2–5, 1962.

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Forty-fifth Annual Meeting: Mark Hopkins Hotel, San Francisco, Calif., April 1–4, 1963.

EDITORIALS

THE SIXTY-THIRD ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Sixty-third Annual Meeting of the American Roentgen Ray Society will be held at the Shoreham Hotel, Washington, D. C., Tuesday, Wednesday, Thursday and Friday, October 2–5, 1962. The Nation's Capital is an ideal place for a meeting of our Society because it is a beautiful and fascinating city and the Shoreham Hotel situated on scenic Rock Creek Park is noted for gracious hospitality offering splendid accommodations. Four times during the past decade, in 1951, 1954, 1957 and 1958, we have met in Washington. Previous meetings have been so well attended that the Society is returning with much enthusiasm. The success of these meetings has been due in no small measure to the friendly reception given to us by the Washington radiologists and the splendid cooperation of the Committee on Local Arrangements. This year, again serving in the same capacity as they have at the two last Annual Meetings in Washington, Dr. Edgar M. McPeak is Chairman and the Ladies Committee is under the able direction of Mrs. Edgar M. McPeak. To those attending is promised a warm reception and delightful entertainment. The Society is most grateful to Dr. and Mrs. McPeak and their Committee Members who have spared no time or effort in planning and organizing the many local details.

All of those who plan to attend are urged to make their reservations immediately with the Shoreham Hotel, Washington, D. C., in order to obtain the accommodations they desire.

The Preliminary Program of the meeting will be found elsewhere in this issue of the JOURNAL. Registration begins at 2:00 P.M. on Sunday, September 30 to 5:00 P.M. and thereafter daily from 8:00 A.M. to 5:00 P.M. except Friday when closing time will be 1:30 P.M. The registration desk will be in the Upper Lobby.

The Annual Golf Tournament will be held at the Manor Country Club, Monday, October 1st. Once again we are deeply indebted to Dr. Ralph M. Caulk for obtaining permission from the Board of Governors for us to be his guests at this beautiful club. Those who have played this fine course will recall the challenge it offers. The clubhouse facilities are most attractive and a delicious dinner and lunch are assured. Year after year this is a wonderful party. Whether you play golf or not, you are urged to attend the luncheon and dinner, relax on the porch and visit. Following the dinner, the Willis F. Manges Trophy for the Eighteen Hole Medal Handicap, and the Exhibitors' Trophy for Eighteen Hole Gross Score with other prizes will be awarded. Colored movies of last year's and previous years' tournaments taken by Arthur Fuchs and his Eastman Kodak associates will be shown. Transportation by buses will be provided to and from the Manor Country Club.

The Scientific Sessions will be held in the Terrace Banquet Room Tuesday through Friday beginning at 8:30 A.M. and ending at 12:30 P.M. This provides ample time to attend the Instruction Courses in the mid-afternoon, for visits to the Technical Exhibits until 5:00 P.M. and the Scientific Exhibits until later in the evening.

The Annual Meeting will be called to order by President Traian Leucutia at 8:30 A.M. Tuesday, October 2nd. The Address of Welcome will be given by the Honorable Walter N. Trobriner, President

of the Board of Commissioners of the District of Columbia, after which Dr. Robert C. Pendergrass, Chairman of the Executive Council, will assist Dr. Traian Leucutia in the installation of President-Elect Earl E. Barth as the new President of the Society. Following the Inaugural Address of President Barth, the Scientific Sessions will begin.

The Program Committee has selected 44 papers to be presented at the Scientific Sessions. A timely and important symposium on "The Role of the Radiologist in Fallout Problems," moderated by Dr. James P. Cooney will be presented at the Thursday session. In keeping with the interest expressed at the last meeting in Miami Beach, the hour 1:30 P.M. to 2:30 P.M. on Wednesday will be devoted to a panel discussion of "Some Activities of the American College of Radiology—1962" moderated by Dr. Arthur J. Present, President of the College.

The format of the Sessions has been altered somewhat this year in that papers in the field of Therapy will be presented each day interspersed with those pertaining to Diagnosis. An attempt has been made by the Program Committee to select topics of general interest. A wide variety of subjects will be discussed by many outstanding radiologists.

On Tuesday morning the papers will be devoted to the examination, diagnosis and therapy of intracranial disorders, both benign and malignant.

The Caldwell Lecture, a notable event at all Annual Meetings, will be given Tuesday evening, October 2nd, in the Terrace Banquet Room at 8:15 P.M. by Dr. C. Allen Good, Head, Section of Diagnostic Roentgenology, Mayo Clinic, Rochester, Minnesota, and well known to members as the untiring Secretary of the Society. He has chosen as his subject, "Tumors of the Small Intestine." Needless to say, this will be one of the highlights of the meeting as no one is more qualified to discuss this subject than our Caldwell Lecturer. Dr. Good will be introduced by a previous associate, long-time close friend, and a distinguished member of the Society, Dr. Clyde A. Stevenson, Director of Radiology, Sacred Heart Hospital, Spokane, Washington. The Caldwell Medal will be presented to Dr. Good at the conclusion of his lecture.

Wednesday morning's program will be concerned with the gastrointestinal tract including one paper related to therapy and one presentation pertaining to radiobiology. On Thursday the papers selected will deal with abnormalities of the chest and great vessels, mammography, isotopes, lymphangiography and dosimetry. The last hour will be devoted to a symposium on the significant subject—"The Role of the Radiologist in Fallout Problems."

The program on Friday pertains to the genitourinary tract and pelvis. Included in this excellent group of presentations are papers dealing with numerous special procedures such as nephrotomography, renal angiography, aortography, cine fluorography, intra-osseous venography, lymphangiography and therapy. This session promises to be of unusual interest and therefore will merit consideration by all attending the meeting.

The Director of Instruction Courses, Dr. Harold O. Peterson, assisted by Dr. Donn G. Mosser, Associate Director, as in the past, has assembled a fine faculty, retaining many outstanding instructors and recruiting a number of new faculty members. An excellent group of courses is offered. An Editorial and the detailed curriculum of the courses with a list of the members of the Faculty will be published in the August issue of the JOURNAL.

The Committee on Scientific Exhibits, of which Dr. James P. Cooney is the Chairman, reports that the Scientific Exhibits will be of the usual high caliber and that the ever-continuing advances in all fields of the specialty will be adequately covered. Special procedures described by several of the essayists as well as several other papers will be supplemented by scientific exhibits.

The number of Technical Exhibits promises to be one of the largest the Society

has had, and probably the largest it has ever had in Washington, D. C. as reported by Dr. James C. Cook, Manager of the Annual Meeting, and Mr. Clifford L. Sherratt, Honorary Member. The Exhibits will be housed in the Main Ballroom—West Lobby Foyer, Bird Cage Walk and Lower Foyer. These Technical Exhibits represent an indispensable part to the success of the Annual Meeting, providing visual documentation of the latest accomplishments in the technical field.

The meetings in Washington have always been popular with the ladies, due not only to the fine facilities but in a larger part to the excellent programs arranged by the Ladies Committee. This year, the Ladies Committee under the able and enthusiastic Chairmanship of Mrs. Edgar M. McPeak has arranged a program which promises to surpass in interest those of the previous meetings. On Monday, for the ladies, guests and those who do not attend the Golf Outing, there will be dinner at a famous seafood restaurant, followed by a night sightseeing tour of Washington. An Embassy Tea and Tour has been scheduled for Tuesday. On Wednesday there will be a luncheon followed by a new and most original program. The always popular and interesting tour of the White House, Mount Vernon, and Arlington with luncheon in Alexandria, Virginia, is planned for Thursday, October 4th. Friday is a free day which the ladies can devote to shopping.

The Annual Banquet will be held at 7:30 P.M., Thursday evening in the Terrace Room. It will be preceded by a cocktail party at 6:30 P.M. to 7:30 P.M. in the Lower Lobby. Scientific awards and the usual introductions are scheduled but no "speeches." Dr. McPeak and his Committee have arranged a program of delightful entertainment which will be followed by dancing. With dinner music, excellent entertainment and dancing, everything is set for a gala evening.

A cordial invitation is extended not only to the Members of the Society, but to all radiologists, their families and guests to attend the Sixty-third Annual Meeting in the beautiful city of Washington, D. C.

EARL E. BARTH, M.D.
President-Elect

670 N. Michigan Avenue
Chicago 11, Illinois



IVAN HAMILTON SMITH, M.D., M.Sc.
1904-1962

AN APPRECIATION

THE sudden and untimely death of Dr. Ivan Hamilton Smith on Saturday, April 7th, 1962 removed an internationally recognized authority in the field of cancer therapy. He had just returned to his home after attending the meeting of The American Radium Society in New York. When a man like Ivan Smith dies, something is irretrievably lost which cannot be expressed in mere words. However, the memory he leaves with those who knew him well, who worked with him, and the great number

who benefited from his skill, his wisdom and his kind heart will attest to his attainments and the place he earned in his profession and the life of the community in which he lived.

Born near Carman, Manitoba, Ivan Smith was the son of a Presbyterian minister. Raised in the simple environment of a rural community, he developed a true sense of values and an appreciation of the needs and plights of his fellow man. No doubt it also engendered his love for the great outdoors. He was an ardent fisherman, gardener and naturalist. He derived much pleasure and refreshment in body and mind from these pursuits and his close association with nature.

He attended public schools in both Manitoba and the London and Dorchester districts. Most of his high school education was obtained in Manitoba but was completed in London. He graduated in medicine at The University of Western Ontario in 1927 and after spending two years' in pathology and obtaining his Master of Science degree in 1928, he went to the Howard A. Kelly Hospital, Baltimore, where he trained under Dr. Kelly and Dr. Thomas Cullen. Kelly, a gynaecologist, was one of the early pioneers in the use of radium in the treatment of human cancer, and it may well be that the spark of interest in radiotherapy was kindled in Ivan Smith's mind at that time. His interest in pathology prompted him to serve as hospital pathologist for the next four years in Parkersburg, West Virginia. Then with renewed enthusiasm he continued his postgraduate education in Edinburgh, receiving his surgical training under Sir David Wilkie, which led to his F.R.C.S. (Edin.) in 1935. Following this, he was resident and assistant in radiotherapy at the Christie Hospital and Holt Radium Institute in 1936–37. By this time the path toward his life's work lay clearly before him and he spent six months visiting cancer centres in Paris, Hamburg, Brussels, Copenhagen and London before he returned home to join the Department of Radiotherapy at the Victoria Hospital, under Dr. George McNeill and later Dr. William Bryan whom he succeeded as Director of the Ontario Institute of Radiotherapy in London. Ivan Smith's enthusiasm and the whole-hearted support of The Ontario Cancer Treatment and Research Foundation resulted in the building of the new quarters for the Cancer Clinic at the Victoria Hospital. Under his guidance, a small department of radiotherapy grew to be the second largest radiotherapy centre in Ontario, and in addition, as the result of his foresight, a well organized research centre developed. As Director of this important unit of the Ontario Cancer Foundation, he served the final years of a distinguished career.

Ivan Smith was a real pioneer in the use of cobalt beam therapy. Commencing in October, 1951 at the London Clinic, he was the first in the world to use cobalt radiation in the treatment of human cancer. In terms of sheer personal experience and practical knowledge, from success and failures, triumphs and disappointments, from learning the hard way, few people knew more than he about radiotherapy. He became an international authority on cobalt radiation. From far and near, his advice and guidance were sought. He was not merely a radiotherapist, but from his training and practice a cancer therapist in the widest sense of the word. Combined with his surgical knowledge, he learned from the colleagues whom he attracted to his Clinic the basic principles upon which the newer and progressive field of chemotherapy is developing. It must have been a constant source of pride for him to see the capable, loyal and devoted staff which served under his inspired leadership.

Finally, in 1961 he had a long time ambition gratified. Approval was given to form a new Department of Therapeutic Radiology in the Faculty of Medicine of The University of Western Ontario and he became its first Professor and Head. This gave him great satisfaction, as it signified the status and prestige which he and the

London Clinic had attained. It is unfortunate that he was not spared a longer span of life to reap more abundantly the fruits of his labours.

In the midst of the constant demands of his professional activities, Ivan Smith was able to give unstintingly of his time and efforts to further the interests and development of the Canadian Cancer Society. He played a major role in its growth and over the years served on the Grand Council for Canada, and the Board of Directors and many committees of the Ontario Division of the Society.

Naturally his honours and associations were many. He was a Fellow of the American College of Radiology and of the International College of Surgeons. He was a member of the American Radium Society, the Radiological Society of North America, the Canadian Association of Radiologists, and the New York Academy of Sciences. He was an honorary member of the Seattle Academy of Surgery, the Radiology Society of the Argentine and the Academy of Medical Sciences Andra Pradesh, Hyderabad, Deccan, India. His greatest honours were, however, the high esteem of his colleagues and the grateful affection of many sick persons who sought his help.

W. G. Cosbie, M.B.

PRELIMINARY PROGRAM

SIXTY-THIRD ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Sixty-third Annual Meeting of the American Roentgen Ray Society will be held at the Shoreham Hotel, Washington, D. C., Tuesday through Friday, October 2 to 5, 1962.

The Executive Council will meet Sunday, September 30, 9:00 A.M. in the Green Room.

On Monday, October 1, the Annual Golf Tournament for Members and Guests—competing for the Willis F. Manges Trophy and the Exhibitors' Trophy—will be held at the Manor Country Club, followed by the Annual Golf Dinner at 7:30 P.M. at the Club.

The Scientific Sessions will be held in the morning from 8:30 A.M. to 12:30 P.M., Tuesday through Friday in the Terrace Banquet Room. The Caldwell Lecture will be delivered by Dr. C. Allen Good on Tuesday evening at 8:15 P.M. in the Terrace Banquet Room. Dr. Good will be introduced by Dr. Clyde A. Stevenson.

The Instruction Courses will be held in the afternoon from 3:00 P.M. to 4:30 P.M., Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M., Friday. The detailed plan of the Instruction Courses will be published in the August issue of the JOURNAL.

The Scientific Exhibits will be set up in the West Ballroom and Foyer. They will open daily at 8:00 A.M. and will close evenings at 8:00 P.M. Tuesday, 9:00 P.M. Wednesday, and 7:30 P.M. Thursday.

The Technical Exhibits will be located in the Main Ballroom, West Lobby Foyer, Bird Cage Walk and Lower Foyer. They will open at 8:00 A.M. and will close at 5:00 P.M., Tuesday through Thursday.

Both the Scientific Exhibits and Technical Exhibits will remain open Friday from 8:00 A.M. until 1:00 P.M.

The Annual Banquet will be held Thursday evening at 7:30 P.M. in the Terrace Room, preceded by a Cocktail Hour beginning at 6:30 P.M. in the Lower Lobby.

The Program Committee under the Chairmanship of President-Elect Earl E. Barth has arranged the following program for the Scientific Sessions.

Tuesday, October 2, 1962
Terrace Banquet Room

8:30 A.M. Call to Order, Sixty-third Annual Meeting: Traian Leucutia, M.D., Detroit, Michigan, President. Address of Welcome: The Honorable Walter N. Tobriner, President of the Board of Commissioners of the District of Columbia.

Installation of President-Elect, Earl E. Barth, M.D., Chicago, Illinois by Traian Leucutia, M.D., President, Detroit, Michigan and Robert C. Pendergrass, M.D., Americus, Georgia, Chairman of the Executive Council.

Inaugural Address: President Earl E. Barth, M.D.

9:10 A.M.

Presiding: Earl E. Barth, M.D.
Chicago, Illinois

1. Normal and Pathologic Roentgen Anatomy of the Middle Ear and Mastoid Process. Lewis E. Etter, M.D., Pittsburgh, Pa.
2. The Roentgenographic Manifestations of Tumors of the Glomus Jugulare. Reed P. Rice, M.D. (by invitation), Chapel Hill, N. C., and Colin B. Holman, M.D., Rochester, Minn.
3. Polytomography of the Ear: Demonstration of Pathologic Conditions. Galdino E. Valvassori, M.D. (by invitation), Chicago, Ill.
4. Simple Radiographic Demonstration of Eustachian Tubes and Abnormalities. M. H. Wittenborg, M.D., Boston, Mass., and E. B. D. Neuhauser, M.D., Boston, Mass
5. Neuroradiologic Potpourri in Infants and Children. E. F. Van Epps, M.D., Iowa City, Iowa.
6. Neoplasms of the Palatine Arch. Mi ford D.

Schulz, M.D., Boston, Mass., Donald Lintner, M.D. (by invitation), Boston, Mass., and Lawrence Sweeney, M.D. (by invitation), Boston, Mass.

7. Dosage to Important Sites: Radiation Therapy of Tumors About the Head and Neck. Robert G. Parker, M.D. (by invitation), Seattle, Wash., Peter Wooton, Hon.B.Sc. (by invitation), Seattle, Wash., and Leland L. Burnett, M.D. (by invitation), Seattle, Wash.
8. Routine Skull Roentgenography of Psychiatric Hospital Admissions. E. Kraft, M.D., Northport, N. Y., N. Finby, M.D., Northport, N. Y., and A. A. Schillinger, M.D. (by invitation), Northport, N. Y.
9. Angiographic Differential Diagnosis of Unilateral and Bilateral Subdural Hematomata. Sidney W. Nelson, M. D., Columbus, Ohio.
10. Exophthalmos Response During X-ray Therapy. Robert J. Blahut, M.D. (by invitation), Ann Arbor, Mich., William H. Beierwaltes, M.D. (by invitation), Ann Arbor, Mich., and Isadore Lampe, M.D., Ann Arbor, Mich.

Tuesday Evening, October 2, 1962
Eight-fifteen o'clock
Terrace Banquet Room

THE CALDWELL LECTURE

Tumors of the Small Intestine

by

C. Allen Good, M.D., M.S. (Radiol.)

Head, Section of Diagnostic Roentgenology, Mayo Clinic and Professor of Radiology, Mayo Foundation Graduate School, University of Minnesota, Rochester, Minn.

Introduction:

Clyde A. Stevenson, M.D., Director of Radiology, Sacred Heart Hospital, Spokane, Wash.

Wednesday, October 3, 1962
Terrace Banquet Room

8:30 A.M.

Presiding: Richard Schatzki, M.D.
Belmont, Massachusetts

11. The Duodenal Loop. Fred O. Coe, Washington, D. C., Charles E. Bickham, Jr., M.D., Washington, D. C., and Carolyn Edwards, M.D. (by invitation), Washington, D. C.
12. The Roentgen Findings in Strictures of the Colon Associated with Ulcerative and Granulomatous Colitis. Richard H. Marshak, M.D., New York, N. Y., Claude Bloch, M.D. (by invitation), New York, N. Y., and Bernard S. Wolf, M.D., New York, N. Y.
13. Perigastric Adhesions—The 'Trapped-Air' Sign. Charles E. Shopfner, M.D. (by invitation), Grand Junction, Colo.
14. Correlations Between Cholangiography and Biliary Tract Endoscopy. Clarence J. Schein, M.D. (by invitation), New York, N. Y., Wilhelm Z. Stern, M.D. (by invitation), New York, N. Y., Elliott S. Hurwitt, M.D. (by invitation), New York, N. Y., and Harold G. Jacobson, M.D., New York, N. Y.
15. Roentgen Manifestations in Intrahepatic Biliary Atresia. David H. Baker, M.D. (by invitation), New York, N. Y., and Ruth C. Edwards, M.D. (by invitation), New York, N. Y.
16. Biplane Gastroduodenal Examination—A New Approach Using the Effect of Gravity on Mobility. Harold M. Landsman, M.D. (by invitation), Little Rock, Ark., and Howard J. Barnhard, M.D., Little Rock, Ark.
17. The Roentgen Manifestations of Hypokalemia. Robert M. Lowman, M.D. (by invitation), New Haven, Conn., Sanford M. Mossberg, M.D. (by invitation), New Haven, Conn., Seymour Haber, M.D. (by invitation), Baltimore, Md., and Barry T. Held, M.D. (by invitation), New Haven, Conn.
18. Radiation Therapy with High-Energy Electrons Using Pencil Beam Scanning. Part II. Clinical Experience, 5 to 40 Mev. J. W. J. Carpender, M.D., Chicago, Ill., Lester S. Skaggs, Ph.D. (by invitation), Chicago, Ill., L. H. Lanzl, Ph.D. (by invitation), Chicago, Ill., and M. L. Griem, M.D. (by invitation), Chicago, Ill.
19. A Study of the Effects of Barium and Intestinal Contents on the Peritoneal Cavity. Donald Q. Cochran, M.D. (by invitation), Columbia, Mo., Carl H. Almond, M.D. (by invitation), Columbia, Mo., and William A. Shucart, M.D. (by invitation), Columbia, Mo.
20. The Thermoplastic Diagnostic Balloon in

the Gastrointestinal Tract. Galen B. Cook, M.D. (by invitation), St. Louis, Mo., and Alexander R. Margulis, M.D., St. Louis, Mo.

21. Diagnostic Problems of Esophageal Cancer. William Seaman, M.D., New York, N. Y., Josephine Wells, M.D. (by invitation), New York, N. Y., and C. A. Flood, M.D. (by invitation), New York, N. Y.
22. Some New Portable Radioactive Sources for Diagnostic Radiography. Kenneth L. Krabbenhoft, M.D., Detroit, Mich., and Farno L. Green, M.S. (by invitation), Detroit, Mich.
23. Further Studies on the Oncylosis—Promoting Humoral Substances Produced by Whole Body Irradiation. Melvin Sikov, Ph.D. (by invitation), Detroit, Mich., and James E. Lofstrom, M.D., Detroit, Mich.

Wednesday, October 3, 1962
Terrace Banquet Room

1:30 P.M.

Presiding: Earl E. Barth, M.D.
Chicago, Illinois

Panel: Some Activities of the American College of Radiology—1962

Moderator: Arthur J. Present, M.D., Tucson, Arizona

Panelists: T. J. Wachowski, M.D., Wheaton, Illinois
George Cooper, Jr., M.D., Charlottesville, Virginia
Wallace D. Buchanan, M.D. South Bend, Indiana
John D. Reeves, M.D., Gainesville, Florida
D. Alan Sampson, M.D., Ardmore, Pennsylvania

Thursday, October 4, 1962
Terrace Banquet Room

8:30 A.M.

Presiding: Robert C. Pendergrass, M.D.
Americus, Georgia

24. Radiological Study of Speech. Earl R. Miller, M.D. (by invitation), San Francisco, Calif., Harry Blackfield, M.D. (by invitation), San Francisco, Calif., John Owsley, M.D. (by invitation), San Francisco, Calif., and Lucie Lawson, Ph.D. (by invitation), San Francisco, Calif.
25. Roentgenologic Features of Single and Multiple Coarctations of the Pulmonary Artery and Branches. Brit B. Gay, Jr., M.D, Atlanta, Ga., Robert H. Franch, M.D (by invitation), Atlanta, Ga., Wade H. Shuford, M.D. (by invitation), Atlanta, Ga., and James V. Rogers, Jr., M.D, Atlanta, Ga.
26. Angiography of the Systemic Pulmonary Circulation. John R. Williams, M.D. (by invitation), Dallas, Texas, W. Curtis Wilcox, M.D. (by invitation), Dallas, Texas, William F. Miller, M.D. (by invitation), Dallas, Texas, and Frederick J. Bonte, M.D., Dallas, Texas.
27. Roentgen Diagnosis of Complications of Cystic Fibrosis. Arthur S. Tucker, M.D., Cleveland, Ohio.
28. Evaluation of the Reproducibility of Mammography. Robert L. Egan, M.D., Houston, Texas.
29. The Effects of Radiations on the Physiology of Small Blood Vessels. William T. Moss, M.D., Chicago, Ill., and Sandra Gold (by invitation), Chicago, Ill.
30. Lymphatic Dynamics in Certain Abnormal States. Sidney Wallace, M.D. (by invitation), Philadelphia, Pa., Laird Jackson, M.D. (by invitation), Philadelphia, Pa., Gerald D. Dodd, Jr., M.D., Philadelphia, Pa., and Roy R. Greening, M.D, Philadelphia, Pa.
31. Analysis of the In-Phantom Dosimeter. Majic S. Potsaid, M.D. (by invitation), Boston, Mass.
32. A New Approach to Radiation Dosimetry in Diagnostic Radiology. Russell H. Morgan, M. D., Baltimore, Md.

33. Symposium: The Role of the Radiologist in Fallout Problems

Moderator: James P. Cooney, M.D.
New York, N. Y.

Participants:

Colonel James T. Brennan, MC, U. S. Army, Director, Armed Forces Radiobiology Research Institute (by invitation).
Lauriston S. Taylor, Ph.D., Chief, Radiation Physics Division, National Bureau of Standards.
Colonel James B. Hartgering, MC, U. S. Army, Technical Assistant, Office of the Special Assistant for Science and Technology, The White House, (by invitation).

Friday, October 5, 1962
Terrace Banquet Room

8:30 A.M.

Presiding: Ralph M. Caulk, M.D.
Washington, D. C.

34. Renal Cyst Puncture—A Roentgenologically Controlled Technique. Harold O. Peterson, M.D., Minneapolis, Minn., and John A. Tobin, M.D. (by invitation), Minneapolis, Minn.
35. Xanthomatous Pyelonephritis. Marvin J. Friedenburg, M.D. (by invitation), St. Louis, Mo., and Harlan J. Spjut, M.D. (by invitation), St. Louis, Mo.
36. The Calcaneal Crescent—A Pyelographic Sign of Severe Hydronephrosis. J. S. Dunbar, M.D., Montreal, Que., Can., M. B. Nogrady, M.D. (by invitation), Montreal, Que., Can., and D. W. MacEwan, M.D. (by invitation), Montreal, Que., Can.
37. An Evaluation of Nephrotomography in Urologic Diagnosis. David M. Witten, M.D. (by invitation), Rochester, Minn., and L. F. Greene, M.D. (by invitation), Rochester, Minn.
38. Percutaneous Left Brachial Aortography. Bill G. Karras, M.D. (by invitation), Chicago, Ill., and Abram H. Cannon, M.D. (by invitation), Chicago, Ill.
39. Selective Renal Angiography. Peter M. Tillotson, M.D. (by invitation), New York, N. Y., and Mordecai Halpern, M.D. (by invitation), New York, N. Y.
40. Cinefluorographic Investigation of Genitourinary Tract Function. Theodore A. Tristan, M.D. (by invitation), Philadelphia, Pa., and John J. Murphy, M.D. (by invitation), Philadelphia, Pa.
41. Intra-osseous Venography; Its Value in the Study of Pelvic Malignancy. Robert E. Wise, M.D., Boston, Mass., Ferdinand A. Salzman, M.D., Boston, Mass., and David O. Johnson, M.D. (by invitation), Boston, Mass.
42. Lymphangiography. Patrick A. Dolan, M.D. (by invitation), Houston, Texas.
43. Can Cancer of the Uterine Fundus be Eradicated with Radium Therapy? Charles L. Martin, M.D., Dallas, Texas, and James A. Martin, M.D. (by invitation), Dallas, Texas.
44. Carcinoma of the Ovary. Antolin Raventos, M.D., Philadelphia, Pa., George C. Lewis, Jr., M.D. (by invitation), Philadelphia, Pa., and Joseph Chidiac, M.D. (by invitation), Philadelphia, Pa.
45. An "After-Loading" Cesium Applicator for Carcinoma of the Cervix. H. Horwitz, M.D. (by invitation), Cincinnati, Ohio, and B. K. Bahr, M.S. (by invitation), Cincinnati, Ohio.

NEWS ITEMS

NATIONAL ACADEMY OF SCIENCES—NATIONAL RESEARCH COUNCIL
Awards in Radiologic Research and Nuclear Medicine
James Picker Foundation

On behalf of the James Picker Foundation, the National Academy of Sciences—National Research Council is accepting applications for fellowships and grants in the field of radiology and nuclear medicine for the year 1963–1964. Awards are made in four categories:

The *Advanced Fellowships in Academic Radiology* reflect the primary interest of the James Picker Foundation in the development of personnel for careers in academic radiology. The purpose is to prepare the fellow to meet in full the intellectual demands of an academic position. Emphasis is placed upon the acquisition of a broad background in the basic sciences related to radiology. Preference will be given to applicants under 34 years of age. Completion of clinical training in radiology ordinarily will be a prerequisite. The candidate should be prepared to devote a minimum of 2 years to course work in the basic sciences and to the application of the techniques and methods of these disciplines to radiologic research. In arranging the details of his program, the prospective fellow will have opportunity for consultation with members of the Academy-Research Council's Committee on Radiology. Application will be accepted on nomination by a clinical adviser, who should be a member of the staff of a Department of Radiology in a medical school.

Postdoctoral Research Fellowships are open to recent graduates who desire investigative experience and training in radiology or nuclear medicine. While persons from closely related disciplines are eligible to apply, candidates with some training in radiology and/or nuclear medicine will receive preference. Applicants must hold the M.D., Ph.D., or Sc.D. degree or the equivalent. Awards are for a period of 1 year, but requests for renewal will be considered.

Grants for Scholars are offered to assist medical schools, hospitals, and other research institutions in supporting and developing junior staff members in radiology and nuclear medicine. This is a transitional form of support, designed to bridge the gap between the completion of research fellowship training and the period when the young scientist has thoroughly demonstrated his competence as an independent investigator. The application is submitted by the institution on behalf of the prospective Scholar. If the request is approved, a grant of $6,000 per year will be made directly to the institution as a contribution toward the Scholar's support, or his research, or both. Initial grants are made for 2 years, but renewal for a third year may be recommended.

Grants-in-Aid of Research are designed to encourage investigations in radiology and nuclear medicine offering promise of improvement in methods of diagnosis or treatment of disease. In line with the interests of the Foundation, the program is oriented toward, but not necessarily limited to, the diagnostic aspects of these fields. Radiologic studies involving the use of radioisotopes fall within the scope of this program. Research grants are awarded to institutions, rather than to individuals. Initial awards may be made for 2 years.

Applications in these four categories will be reviewed by the Committee on Radiology of the Academy—Research Council's Division of Medical Sciences. Final determination of awards is made by the Foundation upon recommendation of the Division. Support is not restricted to citizens of the United States or to institutions within this country.

Applications for the fiscal year 1963–

1964 should be submitted by October 1, 1962. Application blanks and further details may be obtained from the *Committee on Radiology, National Academy of Sciences—National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D.C.*

The Medical Research Council of Canada serves as scientific adviser to the James Picker Foundation with respect to its Canadian program. If the proposed studies are to be carried out in Canada, requests for information and application forms should therefore be directed to the Secretary, Medical Research Council, National Research Building, 100 Sussex Drive, Ottawa 2, Canada.

INSTRUMENTAL AND CHEMICAL TECHNIQUES OF ACTIVATION ANALYSIS

The Oak Ridge Institute of Nuclear Studies, which has been active for some 15 years in the training of scientists in the use of radioisotopes, is announcing a special course to be presented September 24–October 5, 1962.

This special course is designed for the scientist or engineer who has already done some work involving radioisotope techniques and is now interested in applications involving neutron-activation analysis. It will include both lecture and laboratory work covering such pertinent topics as gamma spectroscopy, multichannel analyzers, neutron sources, computer applications, and radiochemical separations.

Prominently featured in the course will be the Special Training Division's new neutron generator—a source capable of producing up to 10^{10} neutrons per second. The device will permit experiments in flux determination, the use of fast or slow neutrons, and analyses of the spectra of extremely short-lived radioisotopes.

Under its contract with the U. S. Atomic Energy Commission, the Special Training Division of ORINS—a nonprofit educational corporation of 38 universities—has provided radioisotope-technique training to more than 4,800 scientists from all 50 states and 58 foreign countries.

Further information about the special course in activation analysis can be obtained by writing Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee.

THE ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

The Twenty-fourth Midsummer Radiological Conference of The Rocky Mountain Radiological Society will be held at the Denver Hilton Hotel, Denver, Colorado, August 16, 17, and 18, 1962.

The guest speakers will be: Dr. John Robert Hodgson, Rochester, Minnesota; Dr. John D. Camp, Los Angeles, California; and Dr. Richard H. Marshak, New York, New York.

An excellent program is being prepared.

For further details please write to: Dr. Raymond W. Hammer, Chairman, Publicity Committee, 808 Republic Building, Denver 2, Colorado.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

DIAGNOSIS AND TREATMENT OF ACUTE RADIATION INJURY. Proceedings of a Scientific Meeting jointly sponsored by the International Atomic Energy Agency and the World Health Organization, Geneva, October 17–21, 1960. Cloth. Price, $8.00. Pp. 425, with illustrations and tables. World Health Organization, Distribution and Sales Unit, Geneva, Switzerland, 1961.

With the aim of gathering information on acute radiation injury, particularly on the biologic effects of large doses of ionizing radiation to the whole body or to substantial portions of it, the International Atomic Energy Agency and the World Health Organization jointly sponsored a Scientific Meeting on the Diagnosis and Treatment of Acute Radiation Injury, in Geneva from October 17 to 21, 1960. The participants were chosen from three groups of radiation workers: those who had experience in the handling of persons accidentally exposed to large doses of ionizing radiation; those engaged in the therapeutic use of large doses of total-body irradiation followed by transplants of bone marrow or other tissues; and those working on related problems in experimental animals.

In view of the exceptional interest of the topics presented in the 21 papers read at the meeting and the broad scope of the discussions that followed, it was thought desirable to publish the Proceedings in the form of a book. The present volume was issued by the World Health Organization on behalf of both sponsoring organizations.

The volume is divided into five Sections. The first and largest Section covers the radiation accidents and the acute radiation syndrome in man, including clinical histories of exposed persons, the assessment, diagnosis and treatment of acute radiation injury, and dosimetric data. Section II is concerned with therapeutic whole-body irradiation and tissue transplantation in man; special consideration is given to the problems of procuring, storing and administering human bone marrow and fetal hemopoietic tissues, to the use of whole-body irradiation as a preliminary to renal homotransplantation, and to the problem of the secondary syndrome. Section III deals with the transplantation of hemopoietic tissues in man and in animals. In Section IV the possible value of certain mercaptoamines in the prophylaxis of radiation sickness is discussed. Finally, Section V contains certain problems for future considerations as related to the effects of whole-body irradiation in man and their modification by experimental and therapeutic means.

Each section is followed by reports of the discussions, which throw into clear relief the issues that are still controversial and provide valuable pointers to further research.

The volume is authoritatively edited, the printing is excellent, and the many diagrams, graphs and figures are splendidly reproduced.

BOOKS RECEIVED

ULTRA-VIOLET AND INFRA-RED ENGINEERING. By Dr. W. Summer, F.Inst.E., A.Inst.P., Hon.F.Phys.A. Cloth. Price, $7.50. Pp. 300, with many figures and tables. Interscience Publishers, Inc., 440 Park Avenue South, New York 16, N. Y., 1962.

RADIOACTIVE ISOTOPES IN MEDICINE AND BIOLOGY. MEDICINE. Second edition. By Solomon Silver, M.D., Attending Physician; Chief, Thyroid Clinic, The Mount Sinai Hospital; Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University, New York. Cloth. Price, $8.00. Pp. 347, with 49 illustrations. Lea & Febiger, Washington Square, Philadelphia 6, Pa., 1962.

ACQUIRED SURGICAL LESIONS OF THE ESOPHAGUS. By Clifford F. Storey, M.D., Private Practice, Thoracic and Cardiovascular Surgery, San Diego, California; Formerly, Clinical Professor of Surgery, College of Medicine, State University of New York at New York; Formerly, Chief of the Surgical Service and Head of the Department of Thoracic Surgery, U. S. Naval Hospital, St. Albans, New York; Portsmouth, Virginia, and others. Cloth. Price, $19.00. Pp. 365, with 181 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1962.

DIE TUBERKULOSE DER ENDOTHORAKALEN LYMPHKNOTEN IM KINDESALTER. By Prof. Dr. Oskár Görgényi-Göttche, Wissenschaftlicher Direktor des Staatlichen Kindersanatoriums, Budapest.

Cloth. Pp. 307, with 121 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1962.

Diagnostik der Geschwulstkrankheiten. By Prof. Dr. Heinrich Bartelheimer, Hamburg; and Priv.-Doz. Dr. Hans-Joachim Maurer, Bonn, Germany. Cloth. Price, $42.00. Pp. 565, with 384 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1962. In the U.S.A. and Canada: Intercontinental Medical Book Corporation, 381 Fourth Avenue, New York 16, N. Y.

Calciphylaxis. By Hans Selye, M.D., University of Montreal, Montreal, Quebec, Canada. Cloth. Price, $25.00. Pp. 552, with 318 illustrations of which many are in color, and 12 tables. The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Ill., 1962.

Moving Field Radiation Therapy. Translated by Elisabeth Farber Lanzl from "Die Bewegungbestrahlung" by Felix Wachsmann and Gunther Barth; adapted and expanded by Lawrence H. Lanzl, Ph.D., Associate Professor of Medical Physics; and James W. J. Carpender, M.D. Professor of Radiology, Department of Radiology and Argonne Cancer Research Hospital, The University of Chicago, Chicago, Illinois. Cloth. Price, $10.95. Pp. 265, with 158 illustrations. The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Ill., 1962.

Quantitative Chemical Techniques of Histo- and Cytochemistry. Vol. 1. By David Glick, Ph.D., Head, Division of Histochemistry, Professor, Department of Pathology, Stanford University Medical School, Palo Alto, California. Cloth. Price, $14.50. Pp. 470, with many illustrations and tables. John Wiley & Sons, Inc., Publishers, Interscience Division, 440 Park Avenue South, New York 16, N. Y., 1962.

Tumors of the Breast; Their Pathology, Symptoms, Diagnosis and Treatment. By Max Cutler, M.D., Associate Attending Surgeon, Cedars of Lebanon Hospital; Surgical Staff of Saint John's Hospital; Chief, Breast Service, Cancer Detection Clinic. Formerly, Director, Chicago Tumor Institute; Consultant, Tumor Clinic; and Director, Cancer Research U. S. Veterans' Administration, Hines, Illinois and Washington, D.C. Cloth. Price, $22.50 Pp. 482, with 235 illustrations and 1 plate in color. J. B. Lippincott Company, East Washington Square, Philadelphia 5, Pa., 1962.

A Radiographic Index. By Myer Goldman, M.B., Ch.B., D.M.R.D., Radiological Registrar, Walton Hospital, Liverpool; Ronald S. Miller, M.R.C.S., L.R.C.P., D.M.R.D., Consultant Radiologist, Wigan and Leigh Hospitals; and David Cope, F.S.R., Superintendent Radiographer, Department of Radiodiagnosis, University of Liverpool, Liverpool, England. Cloth. Price, $3.75. Pp. 77. Williams & Wilkins Company, 428 East Preston Street, Baltimore 2, Md., 1962.

Aktuelle Probleme der Staublungenforschung. By Med.-Direktor Dr. H. Hofmann, Kutzenberg/Lichtenfels-Ofr., Germany. Paper. Pp. 163 with 74 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart N, Germany, 1962. In the U.S.A. and Canada: Intercontinental Medical Book Corporation, New York 16, N. Y.

Flächen- und Elektrokymographie; in der Röntgenologischen Diagnostik der Mediastinal- und Lungenerkrankungen. By Priv.-Doz. Dr. Josef Lissner, Röntgenabteilung der I. Med. Universitätsklinik, Frankfurt/Main, Germany. Paper. Price, $1.40. Pp. 112, with 86 illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart N, Germany, 1962. In the U.S.A. and Canada: Intercontinental Medical Book Corporation, New York 16, N. Y.

La Trasposizione Dei Grossi Vasi; Studio Radiologico. By Franco Fossati, Franco Barbaccia, and Giuseppe Pompili. Paper. Pp. 213, with 102 illustrations. Minerva Medica, 1961.

Effects of Ionizing Radiation on the Nervous System. Proceedings of the Symposium sponsored by The International Atomic Energy Agency and held at Vienna, June 5–9, 1961. Paper. Pp. 509, with many illustrations. International Atomic Energy Agency, Vienna, 1962.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Shoreham Hotel, Washington, D. C., Oct. 2–5, 1962.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Justin J. Stein U.C.L.A. Medical Center, Los Angeles 24, Calif. Annual meeting: Mark Hopkins Hotel, San Francisco, Calif., April 1–4, 1963.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb.
Treasurer, Dwight Vincent Needham, 713 E. Genessee St., Syracuse, N. Y. Annual meeting: Palmer House, Chicago, Nov. 25–30, 1962.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 6–9, 1963.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: Atlantic City, N. J., June 16–20, 1963.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. The Fall 1962 examination will be held at the Pioneer Hotel, Tucson, Arizona, the first week in December; the deadline for filing applications was July 1, 1962. Please note that at this session Nuclear Medicine will become a mandatory part of the examination. All candidates, excluding re-examinees, who are applying for examination in Radiology or Therapeutic Radiology for the December, 1962 session or thereafter *must submit a Nuclear Medicine application* in addition to their basic application. This applies even to those applicants whose basic applications are on file but who will not yet have appeared for examination.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

TENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

EIGHTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colo. Meeting to be announced.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. Don E. Matthieson, 926 East McDowell Rd., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary, Dr. Herbert M. Stauffer, Temple University Medical Center, Philadelphia 40, Pa.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bernard J. Ostrum, 2412 North 52nd St., Philadelphia, Pa.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. Leslie L. Alexander, 257 New York Ave., Brooklyn 16, N. Y. Meets first Thursday of each month October through May.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Edward A. Dunlap, Jr., 35 Danbury Lane, Kenmore 17, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Edward W. Carsky, Crouse-Irving Hospital, 820 S. Crouse Ave., Syracuse, N. Y. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Dana R. Schmidt, 1500 West Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Abram Cannon, Chicago Wesley Memorial Hospital, 250 E. Superior St., Chicago 11, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Ward D. Heinrich, Huron Road Hospital, Cleveland 12, Ohio. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. Seward Imes, 1845 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. James L. Krieger, 85 Jefferson St., Hartford 6, Conn. Meets first Friday in February and April.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary, Dr. Robert D. Moreton, 816 Medical Arts Bldg., Fort Worth, Tex. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. Arch H. Hall, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Bldg., Knoxville, Tenn. Meets in January and September.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Eastern Conference of Radiology
Secretary, Arrangements Committee, Dr. Bernard S. Wolf, The Mount Sinai Hospital, 11 E. 100 Street, New York 29, N. Y. Annual meeting to be announced.

Eastern Radiological Society
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society
Secretary, Dr. Richard D. Shapiro, 1680 Meridian Ave., Miami Beach, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1800 Druid Rd., Clearwater, Fla.

Georgia Radiological Society
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. T. Hunter Pryor, Jackson Memorial Hospital, Miami 36, Fla. Meets monthly, third Wednesday, at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary, Dr. William E. Powers, St. Louis, Mo.

Houston Radiological Society
Secretary, Dr. W. E. Kirkpatrick, 2708 Weslayan, Houston 27, Tex. Meets last Monday each month in fall winter and spring, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.
Secretary, Dr. Richard A. Silver, 712 Hume Mansur Bldg., Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Roger K. Wallace, Riley County Hospital, Manhattan, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Lawrence A. Davis, 226 East Chestnut St., Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. Abraham Berens, 1313 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Saul Heiser, Los Angeles, Calif. Meets second Wednesday of month in September, November, January, March and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Albert A. Poulin, Thayer Hospital, Waterville, Me. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. Irving K. Ettman, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday of each month September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Abraham Marck, Mayfair Professional Bldg., Milwaukee 13, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Jack K. Goodrich, University Medical Center, Jackson, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. John M. Fritts, 1224 S. Higgins Ave., Missoula, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Herbert Kantor, 71 S. Oyster Bay Rd., Syosset, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska State Radiological Society
Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society
Secretary, Dr. George F. Fraser, Las Vegas, Nev.

New England Roentgen Ray Society
Secretary, Dr. Robert E. Wise, 605 Commonwealth Ave., Boston 15, Mass. Meets third Friday of each month, October through April at The Longwood Towers, Brookline, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Bernard S. Wolf, Mt. Sinai Hospital, New York, N. Y. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. A. B. Croom, 624 Quaker Lane, High Point, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. John Jestadt, Depuy-Sorkness Clinic, Jamestown, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Lester I. Citrin, St. Mary's Hospital, Troy, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Northwestern Ohio Radiological Society
Secretary, Dr. Anthony F. Lalli, 421 Michigan St., Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. Chapin Hawley, 927 Carew Tower, Cincinnati, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. Simon Pollack, Utica Square Medical Center, Tulsa, Okla. Meets in January, May and October.

Oregon Radiological Society
Secretary, Dr. George R. Satterwhite, Willamette Falls Community Hospital, 15th and Division, Oregon City, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. John N. Burkey, 555 Dental Bldg., Seattle, Wash. Annual meeting: Seattle, Wash.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Frederick R. Gilmore, Clearfield Hospital, Clearfield, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Antolin Raventos, 3400 Spruce St., Philadelphia 4, Pa. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Ross H. Smith, St. Margaret Memorial Hospital, Forty-Sixth St., Pittsburgh 1, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. William C. Duffey, Cincinnati, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. G. J. Liese, Queen's Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Robyn Hardy, 4324 Magnolia St., New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. George H. Burke, 601 Grand Ave., Asbury Park, N. J. Meets in Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Robert Scanlan, St. Vincent's Hospital, Los Angeles, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Harry J. Pinsky, 386 Monroe Ave., Rochester 7, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 16–18, 1962.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Charles G. Campbell, 6673 Avenida Manana, La Jolla, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Walter Coulson, San Francisco General Hospital, San Francisco 8, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Wayne P. Whitcomb, Hospital of St. Raphael, New Haven, Conn. Meetings are held bimonthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1746 K St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. Richard G. Lester, 412 Union St., S.E., Minneapolis 14, Minn. Annual meeting: Shoreham Hotel, Washington, D. C., Sept. 30–Oct. 1, 1962.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. *Administrator*, Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill.

South Bay Radiological Society
Secretary, Northern Section: Dr. John S. Wilson, 1515 Trousdale Dr., Burlingame, Calif.; Southern Section: Dr. A. C. Mitchell, 728 Cass, Monterey, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting: Grand Hotel, Point Clear, Ala., Feb. 1–3, 1963.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Mem-

phis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Tex. Annual meeting: San Antonio, Tex., Jan. 25 and 26, 1963.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Welborn Clinic, 420 Cherry St., Evansville, Ind. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society
Secretary, Dr. Powell G. Dillard, Jr., 715 Church Street, Lynchburg, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Joseph T. Houk, 14303 Ambaum Blvd., Seattle 66, Wash. Meets third Monday of each month from September through April at the University of Washington Medical School.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Edward T. Bello, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2–37, Zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15. México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. F. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César E. Rosa-Perez, Fondo del Segura del Estado, Parada 1, San Juan 8, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Edmonton and District Radiological Society
Secretary, Dr. S. C. Windle, 105 Northgate Bldg., Edmonton, Alberta, Canada. Meets first Tuesday of each month, October to May.

Faculty of Radiologists
Honorary Secretary, Dr. C. J. Hodson, 47 Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. D. J. Sieniewicz, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Que., Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. Wallace M. Roy, St. Joseph's Hospital, 30 The Queensway, Toronto 3, Ont., Canada. Meets second Monday of each month September through May.

College of Radiologists of Australia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut., Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Skeletal System

Isherwood, Ian. Spinal intra-osseous venography. *Clin. Radiol.*, Jan., 1962, *13*, 73–82. (From: Department of Radiology, Manchester Royal Infirmary, Manchester, England.)

Much of our current knowledge of the vertebral veins is due to the work of Oscar V. Batson, who described the role of these veins in the spread of malignant disease. The plexus of veins about the vertebral column may be divided into internal and external groups (inside and outside the vertebral canal). These plexuses anastomose freely. The internal vertebral plexuses lying within the vertebral canal form a close network and run mainly in a vertical direction forming 4 longitudinal veins, 2 in front and 2 behind the spinal cord. Blood from the cancellous tissue of the vertebrae is collected by the basivertebral veins which take a radial direction and communicate with the longitudinal veins. The azygos vein is a continuation of the right ascending lumbar vein. The inferior hemi-azygos is the extension of the left ascending lumbar vein. The superior hemi-azygos descends on the left side of the vertebral column and crosses to enter the azygos at the level of the seventh thoracic vertebra. The azygos and hemi-azygos veins receive the intercostal and many mediastinal veins. The dorsal branches of the intercostal veins receive the intervertebral veins and thereby provide the drainage for the vertebral plexuses to the azygos system. Hence, two systems of veins unite the superior vena cava with the inferior vena cava: (1) lumbo-azygos veins within the body cavity; (2) vertebral veins outside the body cavity in the bony canal of the vertebral column.

Intra-osseous venography. Contrast material when injected into bone marrow is rapidly taken up by the local venous drainage. The vertebral spinous process is an easily accessible bony prominence with rich draining venous plexuses. Postmortem injection of a spinous process results in extensive filling of the vertebral venous plexuses extending into the azygos vein and superior vena cava via the dorsal branches of the intercostal veins.

A preliminary test dose of contrast material is given. Intra-osseous injection is performed under local anesthesia with the patient lying prone on the roentgenographic table. Preliminary lateral and posteroanterior films are exposed. An iliac crest bone biopsy needle, No. 17, with a piece of metal "T" tubing attached to the trochar head to allow some rotary movement is used. The needle is introduced through the cortex by steady pressure and rotation. Its entry into the medulla is easily recognized, and blood should ooze from the needle as if in a vein. A test injection of 5 cc. of 45 per cent hypaque is made, and a lateral film is exposed to establish with certainty the position of the tip of the needle. Twenty cc. of 45 per cent hypaque is then injected as rapidly as possible. Roentgenograms are taken toward the end of the injection, and then at one or two second intervals. Three roentgenograms usually suffice. For the posteroanterior films the injection is repeated.

Clinical studies using intra-osseous venography have been made for: (1) investigation of spinal angiomata; (2) investigation of low back pain and obstructive lesions of the spinal canal; (3) inferior veno-cavography; (4) investigation of portal hypertension; and (5) investigation of diseases of the chest and mediastinum, particularly where abnormal opacities above the right hilus are diagnostic problems. The demonstration of the azygos vein as only a part of such opacity may be helpful in diagnosing mediastinal involvement in a patient with a peripheral bronchogenic carcinoma. Six case studies are presented.—*Samuel G. Henderson, M.D.*

General

Paul, F. M. Tuberculosis in B.C.G. vaccinated children in Singapore. *Arch. Dis. Childhood*, Oct., 1961, *36*, 530–536. (From: The Paediatric Unit, General Hospital, Singapore, Malaya.)

The author reports the results from the use of B.C.G. vaccine on newborn children at the Kandang Kerbau Maternity Hospital in Singapore. The report summarizes 14 cases of tuberculous meningitis and 2 cases of severe pulmonary tuberculosis in children who had received B.C.G. vaccine at birth and had been admitted to the Pediatric Unit of the General Hospital in 1958, 1959, and 1960. Six of these children were in the 2 year age group, 4 were one year old, and 4 under one year; the youngest case was 7 months old. All of the cases had received B.C.G. vaccine at the above named unit. The Mantoux test had not been checked in all of the children. It was felt that the Mantoux test should change to positive within 3 months after injection of B.C.G. vaccine.

Thirteen of the 14 cases had positive Mantoux tests at the time of admission to the hospital. The one case which was negative on admission became positive 3 months after a B.C.G. vaccine at birth. This patient died during the hospital admission. Twelve of the 14 cases had a history of symptoms lasting 10 days or less.

The author noted the hospital course of several cases and he describes the findings in 3 of the cases that died. There were 2 cases which had fulminating pulmonary tuberculosis.

It is concluded that there had been adequate demonstration that B.C.G. vaccine was of good use in preventing tuberculosis. At birth it is a worthwhile procedure but it does not provide absolute immunity in childhood.—*Paul L. Webster, M.D.*

Atta, A. G., and Hoch, J. Marfan's syndrome and dissecting aneurysm of aorta. *A.M.A. Arch. Int. Med.*, Nov., 1961, *108*, 781–788. (Address: South Jersey Medical Research Foundation, Sheridan and Copewood Sts., Camden 4, N. J.)

The authors discuss the findings in Marfan's syndrome, present a case of a 22 year old white male, and briefly review the other members of his family who had varying degrees of this disease.

Marfan's syndrome is a heritable disorder of the connective tissue, transmitted by a dominant gene, which is not sex-linked. The complete syndrome shows most of the following abnormalities: (1) in the skeleton: dolichocephaly, arachnodactyly, highly arched palate, scoliosis, pectus carinatum or excavatum, spina bifida, pes planum or highly arched feet, and double row of teeth; (2) in the eyes: ectopia lentis, shallow anterior chambers, irido-donesis (frequent tremor of the eyes), and retinal detachment; (3) in the cardiovascular system: aneurysms of the aorta, valvular and septal defects, coarctation of the aorta, dissecting aneurysms of other major vessels, aneurysms of the sinuses of Valsalva, and ruptured aortic cusps; and (4) hernias and hypermobility of the joints are frequent.

The case presented is that of a young man who had all the classic signs of Marfan's syndrome, including a dissecting aneurysm of the aorta. He had exertional dyspnea, occasional edema of the extremities, and for 2 years prior to admission had frequent episodes of retrosternal pain radiating to the left arm. However, 6 months prior to admission he was involved in an automobile accident and received a violent blow to the chest from the steering wheel; following this accident his heart symptoms were accentuated. Upon admission he had forceful pulsation in the left carotid artery and diminished pulsation in the right carotid artery. The blood pressure in the right arm was 100/40, in the left arm 210/70, in the right thigh 300/150, and in the left thigh 300/90. A continuous, loud, machinery-like murmur with systolic accentuation was heard over the precordium and especially in the third left intercostal space, near the sternum. Chest roentgenograms showed a markedly enlarged heart and some widening of the ascending aorta. At autopsy, done about 40 days after the initial admission, the heart weighed 990 gm.; most of the enlargement was left ventricular. Just above the aortic ring, within the ascending aorta, was a large, saccular dissecting aneurysm, which measured 8.5 cm. in its greatest transverse diameter. Elastic tissue stain of the aorta demonstrated medial necrosis. (The gross and microscopic findings in the aorta are described in detail in the article.)

Review of the family tree of the authors' patient revealed a total of 14 cases of Marfan's syndrome. Some members of the family showed no evidence of this syndrome; others had apparently only minimal manifestations such as arachnodactyly and highly arched palate. Twelve had ectopia lentis. Both males and females were involved. Five had cardiovascular manifestations; none of these were women. The female members of the family who were affected probably had no cardiovascular involvement because of their more sedentary life. Apparently those tissues of the body which are more subject to the wear and tear of living are prone to develop the most serious manifestations of Marfan's syndrome, *i.e.*, ectopia lentis and aortic aneurysm.

This study confirms the hereditary characteristics of Marfan's syndrome; the transmission apparently occurs through a dominant gene which is not sex-linked.—*Richard J. Noveroske, M.D.*

Arredondo G., Hugo, and Ceballos L, Jorge. Unusual location of mycetoma. *Radiology*, Jan., 1962, *78*, 72–76. (Address: Dr. Jorge Ceballos L, Ave. Insurgentes Sur No. 1023–108, México 18, D. F., México.)

The authors present 5 case reports of mycetoma, also known as "Madura Foot," which occurred in Mexico. In each of these cases the lesions were produced by *Nocardia brasiliensis*. Mycetoma is endemic in tropical and subtropical regions and occurs mostly among farmers, with 81 per cent of patients being males.

Clinically, the affected region shows induration and swelling; numerous fistulas drain serous and bloody exudate or, frequently, a discharge containing yellowish-white grains which have been compared to fish roe. Pain, if any, is slight, and fever is present only if complications due to intercurrent illness occur. Roentgenographically the picture varies according to the stage and extent of the disease and is of two general types: changes in the soft tissues and changes with additional bone involvement. Soft-tissue technique roentgenograms show: (a) an irregular contour of the skin caused by the fistulas, the surface nodules, and the irregular cicatrization; (b) an increase in volume and density of the soft tissue due to edema; (c) intramuscular nodules, nearly always multiple, intermediate in density between soft tissue and bone.

Changes in the bone show laminated periosteal new bone formation, similar to that in periostitis. There may be thick, irregular spicules formed, radiating perpendicularly from the cortex, and also thick layering of periosteal new bone formation, irregular in character, a result of the combination of osteolytic and osteoblastic areas. A combination of these types of bone changes may be present. Sometimes thinning of the cortex is apparent, occasionally with areas of disruption. Irregular osteoblastic and osteolytic zones may replace the normal bone architecture. The joints as a rule are not involved unless extensive disease is present.

The differential diagnosis must include malignant processes and osteomyelitis caused by other agents. The soft tissue involvement in mycetoma helps to distinguish this from other types of osteomyelitis, and the slow development of the mycetomas, in addition to the soft-tissue changes, helps in the differentiation from malignant neoplasms.—*Donald N. Dysart, M.D.*

RADIATION THERAPY

SINNER, WALTHER. Karzinome der männlichen Brustdrüse; Beobachtungen an 27 Fällen; Züricher Erfahrungen 1919 bis 1960. (Carcinoma of the male breast; 27 patients observed in Zürich during the period 1919–1960.) *Strahlentherapie*, Aug., 1961, *115*, 522–547. (From: Röntgendiagnostisches Zentralinstitut und Radiotherapeutische Klinik und Poliklinik der Universität, Zürich, Switzerland.)

In this detailed review of the 27 males with carcinoma of the breast seen in Zürich from 1919 to 1960, the author notes that:

1. Carcinoma of the male breast has increased three-fold in the past several decades. The ratio of male to female patients with carcinoma of the breast is 1 to 89; in some tropical regions this ratio is increased by a factor of 10 so that males have about 10 per cent of the total carcinomas of the breast.

2. Mammary carcinoma appears, on an average, 15 years later in males than in females.

3. The delay between the onset of symptom(s) and seeking medical advice averaged 13.1 months for males, as opposed to 4–5 months for females.

4. The first symptoms included: a mass in the breast; pain and itching locally; perceptible increase in size of a mass in the breast; change in the nipple; discharge from the nipple; and local ulceration.

5. Therapy usually consisted of both surgical (usually radical mastectomy) and irradiation (usually prophylactic postoperative); 45 per cent of the patients survived for 5 years, 35 per cent were alive and free of symptoms at the end of 5 years.—*Henry G. Moehring, M.D.*

LOEVINGER, R., KARZMARK, C. J., and WEISSBLUTH, M. Radiation therapy with high-energy electrons. Part I. Physical considerations, 10 to 60 Mev. *Radiology*, Dec., 1961, *77*, 906–927. (From: The Department of Radiology and the Biophysics Laboratory, Stanford University, Stanford, Calif.)

An investigative program directed toward electron beam radiation therapy with the Mark IV 70 mev. microwave linear accelerator has been in progress at Stanford University since 1954. The authors describe the studies on the physical properties of the electron beam which are of radiologic significance. Illustrations and a description of the general layout of the accelerator, treatment room, and therapy control room are given.

The electrons were accelerated in two sections, after which they passed through a double deflection magnetic system. The electron beam emerged from the linear accelerator in a fixed horizontal direction. After passing through a system of monitors, the electrons were scattered in aluminum to provide a beam of the desired width. Beyond the scatterer the beam was collimated to the desired field size and shape. The first (analyzer) magnet and a slit served to define the electron energy and the energy spread. The second (deflection) magnet separated the electron beam from neutrons and x-rays created at the slit. The magnetic field was monitored at the first magnet by a rotating coil flux meter which was calibrated in terms of electron energy by photoneutron activation thresholds. The transmission ion chamber mounted on the end of the accelerator served only as an indicator of accelerator output for the operating technician and played no role in monitoring therapy irradiation. The second emission monitor was the basic monitor for all therapy and test irradiation. It was never used alone in treatment but always in conjunction with one pair of the ion chamber monitors at the downstream end of the collimator.

Depth-dose measurements were made in water, polystyrene, acrylic plastic, Pressdwood, and Mix D. Unexplained discrepancies between the depth-dose curves measured by different methods were found. The best method for making dose distribution measurements was with a small ionization chamber in a water phantom. Central axis measurements were made with a ferrous sulfate dosimeter. The photographic method of obtaining dose distribution data was the most rapid and gave results in good agreement with other measurement procedures.

A number of problems remain to be solved but electron beams can be regarded as a useful therapeutic modality up to an energy of at least 20 mev. The question of the energy at which the disadvantages begin to outweigh the advantages cannot be answered by analysis of the reported physical data.

For further information, the reader is referred to the original of this excellent paper.—*A. W. Sommer, M.D.*

ZATZ, L. M., VON ESSEN, C. F., and KAPLAN, H. S. Radiation therapy with high-energy electrons. Part II. Clinical experience, 10 to 40 Mev. *Radiology*, Dec., 1961, *77*, 928–939. (From: The Department of Radiology, Stanford University School of Medicine, Palo Alto, Calif.)

The authors report the results of 2 years of clinical experience with high-energy electrons produced by a Mark IV linear accelerator. In a 2 year period from

May, 1958 to April, 1960, 42 lesions were treated with electron beams of 10 to 40 mev. Initially, patients with locally advanced primary lesions or metastases in superficial areas were treated. Subsequently patients with less advanced or deeper lesions were accepted for treatment. Patients were treated on a three-treatment-per-week schedule. In 6 of the 42 lesions, low doses were administered either to study the time-dose relationship or because of poor condition of the patient. In the majority of the remaining 36 cases, the minimum tumor dose was about 6,000 rads in 5 weeks. In every instance, the energy of the electron beam used to treat the lesion was one which delivered a minimum tumor dose which was 90 per cent of the maximum dose delivered to the irradiated field.

The longest follow-up after treatment was 30 months and the shortest 4 months. Because of the short interval and the small number of cases, no statistical assessment is possible. Evaluation is therefore on the basis of radiation reaction and tumor response rather than on control of the local disease. Minimum tumor doses of 6,000 rads delivered in 5 weeks through 10 by 12 cm. neck fields resulted in a skin dose of approximately 5,700 rads. This usually produced a severe dry or moist skin reaction. The late skin changes were similar to those following acute reactions of equal severity produced by roentgen rays. Moderately severe late skin changes were seen in one patient who had received previous roentgen treatment in the areas irradiated with the electron beam. The electron beam appeared to have very little skin-sparing effects compared to megavoltage therapy but a definite advantage of electron therapy over 200 kv.p. roentgen therapy with respect to skin dose was noted. The reactions observed in the mucous membrane of the mouth and pharynx were comparable to those produced by similar doses of roentgen rays. Six thousand rads in 5 weeks produced a confluent mucositis.

In 19 of the 42 patients in this series there was gross disappearance of the tumor in the treated area without evidence of recurrence during the period of follow-up. One patient with an extensive carcinoma of the pharynx with bilateral cervical lymph node metastases is alive 25 months following treatment, with no evidence of tumor but with persistent laryngeal edema.

It is concluded that electron-beam therapy is a useful tool for the treatment of relatively superficial lesions. The use of parallel opposing fields with higher-energy electrons for the treatment of deep lesions probably offers little advantage over present megavoltage therapy techniques.—*A. W. Sommer, M.D.*

Harder, Dietrich, Harigel, Gert, and Schultze, Klaus. Bahnspuren schneller Elektronen; Aufnahmen der Elektronen- und Röntgenstrahlung eines 35-MeV-Betatrons mit einer propangefüllten Blasenkammer. (Tracks of fast electrons; photographs of a propane-filled bubble chamber exposed to the electron and roentgen ray beams of a 35 mev. betatron.) *Strahlentherapie*, May, 1961, *115*, 1–21. (From: Max-Planck-Institut für Physik und Astrophysik, München; und Physikalisches Institut der Universität, Würzburg, Germany.)

Photographs of a liquid propane chamber placed in the electron or roentgen ray beam from a 35 mev. betatron show where energy is absorbed because the absorbed energy produces tiny bubbles of propane gas. The substance of this paper is in the 25 illustrations from such photographs. They illustrate the reason for the shapes of depth-dose curves, particularly for the changes associated with successive increases of energy of an electron beam from 4.6 mev. to 21.2 mev.—*Henry G. Moehring, M.D.*

Schneider, Rudolf. Das Herz bei der Tiefentherapie intrathorakaler Tumoren unter besonderer Berücksichtigung der Telekobaltbestrahlung. (The heart in deep therapy of intrathoracic tumors, with particular reference to telecobalt irradiation.) *Strahlentherapie*, July, 1961, *115*, 377–378. (From: Strahlenklinik und Poliklinik der Universität, Marburg/Lahn, Germany.)

The author concludes from a review of the literature and from observations on 309 patients with malignant intrathoracic tumors as follows:

1. Direct damage from irradiation of the heart is rare. Although the radiation therapy should be planned to keep the heart out of the field of maximum dose, when esophageal, mediastinal or pulmonary tumors lie next to the heart, this is not possible and whatever small risk results from cardiac irradiation can be accepted. (Animal experiments indicate that the depth dose and the size of the field are much more important considerations in producing histologically demonstrable cardiac changes than is the difference between whether the radiation came from a 200 kv. or a cobalt unit.)

2. Pre-existent cardiac damage and associated electrocardiographic changes are no contraindication to radiation therapy. In many instances, cardiac complications which arise during radiation therapy need not cause abandonment of therapy.

3. Indirect cardiac damage from radiation therapy arises most commonly from radiation pneumonitis. Superimposed on pulmonary emphysema, radiation pneumonitis can cause subacute cor pulmonale which may be fatal. Pulmonary fibrosis and other late intrathoracic effects of radiation may also produce indirect cardiac effects and are minimized by decreasing the volume of irradiated lung and by decreasing the dose to any one volume of lung; *e.g.*, by

crossfiring through many fields or by rotation therapy.

4. Patients in poor general or cardiac condition should be irradiated, if at all, only after intensive treatment of the cardiac condition.—*Henry G. Moehring, M.D.*

Laurance, Bernard M. Intracranial complications of leukaemia treated with intrathecal amethopterin. *Arch. Dis. Childhood*, Feb., 1961, *36*, 107–110. (From: Derbyshire Children's Hospital, Derby, England.)

Since the advent of the various chemotherapeutic agents, the peripheral blood and bone marrow in acute leukemia have been controlled over longer periods of time. These agents do not prevent central nervous system infiltrates. The author stresses the treatment of intracranial complications.

Two cases are reported to illustrate the complications. These children were treated with 6-mercaptopurine for 10 and 15 months, respectively, prior to the development of central nervous system changes of leukemia. They were then treated with intrathecal amethopterin with marked improvement of the central nervous system signs. The improvement remained over several months but both children succumbed to the disease process.

It seems reasonable to assume that with the increase in the longevity of children with lymphatic leukemia, more intracranial complications will occur. The author cites the importance of frequent examination of the fundi in children with this disease.

There are 4 illustrations (note is made that 2 of these are mislabeled) and 1 table.—*John L. Gwinn, M.D.*

Albright, Edwin C. Malignant melanoma following bilateral adrenalectomy for Cushing's syndrome. *J. Clin. Endocrin.*, Jan., 1962, *22*, 93–99. (From: Department of Medicine, University of Wisconsin Medical School, Madison, Wisc.)

Bilateral adrenalectomy has been successfully used for a number of years. Recently, Nelson and associates have reported corticotropin-producing tumors of the pituitary following adrenalectomy for Cushing's syndrome. Deep pigmentation of the skin was a constant feature in these patients, and was attributed to increased activity of melanophore-stimulating hormone (MSH) derived from the pituitary.

The author has observed a patient with Cushing's syndrome accompanied by mild cutaneous melanin pigmentation and many benign junctional nevi. Twelve months following adrenalectomy the patient was found to have enlargement of the anterior mediastinal lymph nodes and sclerotic changes in the body of the eleventh thoracic vertebra, indicative of metastatic disease. Biopsy of an enlarged supraclavicular lymph node established a diagnosis of malignant melanoma. During the ensuing 14 months until his death, there occurred progressive enlargement of the mediastinal lymph node masses, invasion of the adjoining lung, and sclerotic involvement of the lumbar spine and pelvic bones. A remarkable clinical feature noted during the last 6 months of life was the generalized intense melanin pigmentation of the skin, which assumed a dark chocolate color. It is noted that this patient showed no clinical evidence of a pituitary tumor by roentgen-ray or visual field examination.

The clinical progress of this patient through four admissions to the hospital is discussed. Photographs and reproductions of a microscopic section and roentgenograms are included.—*Douglas S. Kellogg, M.D.*

Born, Willi, and Hubach, Heinrich. Elektroenzephalographische Veränderungen nach Röntgenepilation des Kopfhaares bei Kindern. (Electroencephalographic changes after roentgen epilation of the scalp in children.) *Strahlentherapie*, July, 1961, *115*, 465–477. (From: Hautklinik und Psychiatrische und Nervenklinik der Universität, Freiburg/Br., Germany.)

Forty-nine electroencephalographic studies on 7 children subjected to roentgen epilation were made from just before the epilation to as long as 9 weeks after. The epilation varied from part to all of the scalp and from single to fractionated dosage. Of the seven $3\frac{1}{2}$ to 7 year old patients, 6 showed abnormal changes in their postepilation electroencephalograms; *e.g.*, general slowing of the basic rhythm. These changes were evident the first day after radiation therapy and then receded stepwise.

The authors count these findings as an expression of particular sensitivity to radiation of cerebral tissue in children, and as such, so minor that their indications for roentgen epilation in suitable patients remain unchanged.—*Henry G. Moehring, M.D.*

RADIOISOTOPES

Coenegracht, J. M., and Dorleyn, M. The distribution of intravenously administered tracer doses of Bi-206 compounds in the human body. *J. belge de radiol.*, 1961, *44*, 485–504. (From: Isotope Laboratory, Ziekenhuis Sint Annadal, Maastricht, Netherlands.)

Distribution of colloids or suspensions introduced intravenously is largely independent of the chemical or physiologic properties of the basic constituents, it being determined almost exclusively by the physiochemical properties of the substances; therefore, the size of the particles is important.

Particles smaller than 2.5 mμ. remain in the bloodstream relatively long; they are removed by the reticuloendothelial system, particularly the spleen and liver, and largely by the bone marrow. Particles of 25 mμ. are apt to be filtered out by the liver. In the

case of larger particles of 100 μ. and more considerable retention occurs in the lungs.

A total of 27 patients was given a suspension of Bi^{206} phosphate, the mean diameter of the charcoal suspension being 5 mμ. A dose of 25–140 μc was given to 25 cases. Two patients with malignancy were given larger doses.

The authors conclude that this preparation is not satisfactory for measurement of hepatic blood flow. In 4 patients with malignancy (lung tumor, malignant reticulosis, and Hodgkin's disease), no radioactivity was detected in the neoplastic tissues.—*David C. Alftine, M.D.*

Cavanagh, Denis, Gilson, Albert J., and Powe, Charles E. Isotopic placentography: an evaluation based upon a study of 50 patients. *South. M. J.*, Dec., 1961, *54*, 1340–1346. (From: The Departments of Obstetrics-Gynecology and Radiology of the University of Miami School of Medicine, Jackson Memorial Hospital, Miami, Fla.)

Isotopic placentography was performed upon 50 obstetric patients. The patient was placed on a comfortable padded table in the supine position. Five microcuries of radioactive iodinated human serum albumen (R.I.S.A.) were injected into an arm vein. Radioactive iodine (I^{131}) has a half life of about 8 days.

A counter-balanced 2 by 2 inch sodium iodide scintillation detector, with a minimum of one and a half inch of lead shielding surrounding the crystal, was used for counting. After allowing about 10 minutes for equilibration, the detector was placed over the xiphisternum. The count over the xiphoid was recorded and all uterine counts were applied to this, using the xiphoid count as 100 per cent. The uterine wall was subdivided into 9 segments. A 20 degree flat field collimator was utilized for this procedure.

All sector counts were expressed as a percentage of the count obtained over the xiphoid process, this being taken to represent the cardiac blood pool. Typical normal and abnormal placenta scans are reproduced and discussed. Two errors found were false positives and no false negatives were noted.—*Eugene J. McDonald, M.D.*

Doehner, Gunther A. New scintigraphic technic for demonstration of beta emitters following intracavitary instillation. *Radiology*, Jan., 1962, *78*, 100–102. (Address: 153 West 11th Street, New York 11, N. Y.)

Radiotherapy for carcinomatous exudates in body cavities, using radioactive isotopes in colloidal solution, has earned a place in clinical cancer management. Radiogold, which emits both beta and gamma radiation, has been widely used because of its chemical and physical properties, particularly the rather uniform precipitation of gold colloid on the serosal surfaces. However, early clinical evaluation of the isotope distribution in body spaces is difficult because of the high radiation exposure to the examiner and scintigraphic demonstration of the distribution is feasible only after a lapse of approximately 5 half-lives or approximately 2 weeks.

Attempts to replace this isotope with one of pure beta emission were achieved with the availability of a stable suspension of chromic (P^{32}) phosphate. The radioactive phosphorus does not emit primary gamma radiation and the distribution of the isotope was generally not considered demonstrable after instillation in a body cavity. However, the secondary irradiation or bremsstrahlung resulting from deceleration of the beta particles is sufficiently intense and penetrating (half-value layer of approximately 1.0 mm. Pb) to be demonstrable by scintigraphic methods. In fact, a scintillation crystal of 1 inch diameter is sufficiently sensitive to register the secondary x-radiation produced by 5×10 mc of radioactive phosphorus distributed over a serous cavity.

In clinical use the author replaces the focusing collimator of a scintillation detector with a cylindrical insert and scintigrams are performed in a supine position. The collimation has only moderate accuracy but is sufficient for the purpose since exact detail is not required. In the two scintigrams presented the outline of radioactivity exceeds the size of the serous cavity only by an inch or so.

The author concludes that, with the described technique, a beta emitter such as chromic (P^{32}) phosphate allows better clinical study than a beta-gamma emitter such as radiogold.—*Edward B. Best, M.D.*

Ecklund, Robert, and Ryan, Robert. Suppression of release of radioactive iodine as a test of thyroid function. *J. Clin. Endocrinol.*, Jan., 1962, *22*, 26–30. (From: The Endocrine Laboratory, Department of Medicine, University of Illinois College of Medicine, Chicago, Ill.)

The suppression of I^{131} thyroidal uptake by the administration of desiccated thyroid or triiodothyronine in euthyroid but not hyperthyroid patients has been used as a thyroidal function test since 1954. Johnson, Solomon and Greer showed that thyroxine and triiodothyronine did not slow the discharge of I^{131} from the thyroids of thyrotoxic patients, but did in euthyroid patients.

The authors studied the effects of triiodothyronine and methimazole in 16 euthyroid and 19 thyrotoxic subjects. The methimazole inhibited the reaccumulation of any released I^{131}. The authors confirmed the findings of Johnson *et al.* The thyroidal release of I^{131} did not follow a single exponential function and was thought to represent the net effect of two or more exponential rates of loss.

A practical test developed from the above study consisted of the administration of a tracer dose of

I^{131} followed in 24 hours by the measurement of the thyroidal I^{131} uptake, then the administration of 75 μg. of triiodothyronine and 120 mg. of methimazole per day for 6 days and a second determination of I^{131} thyroidal activity on the seventh day. Approximately 92 per cent of the I^{131} remained in the thyroid of euthyroid subjects, whereas 49 per cent was retained by the thyroid in thyrotoxic patients.

The authors believe that the release-suppression test is as good as the uptake-suppression test in discriminating between hyperthyroid and euthyroid subjects.—*Charles W. Cooley, M.D.*

Sheline, Glenn E., Lindsay, Stuart, McCormack, Kenneth R., and Galante, Maurice. Thyroid nodules occurring late after treatment of thyrotoxicosis with radioiodine. *J. Clin. Endocrinol.*, Jan., 1962, *22*, 8–18. (From: Departments of Radiology, Pathology, and Surgery, University of California School of Medicine, San Francisco, Calif.)

Thyroid nodules were found in 8 of 256 patients treated with I^{131} with diffuse toxic goiter at the University of California Hospitals, San Francisco, from 1946 to 1953. The thyroid nodules were noted 5 to 14 years after the beginning of I^{131} therapy. Six cases were less than 18 years of age at the time of therapy and 2 were between 25 and 30 years of age. The 8 cases are presented in detail. None of the 8 patients were hypothyroid and none had received exogenous thyroid hormone previous to the discovery of the nodules. Four of the patients with the late nodules, including 1 with a nodule which was probably an early carcinoma, were among the 6 who were 10 years or less of age at the time of treatment. No late nodules were found in the 186 patients over 30 years of age.

The benign nodules observed in this study appeared to originate mainly in hyperplastic regenerating areas of epithelium. Chronic thyroiditis or Hashimoto's disease appeared in 6 cases. The development of thyroid nodules in the 8 cases was similar to that observed in irradiated rat thyroids. No nodules were found in the cases that were hypothyroid after treatment with I^{131}.

The nodules that develop after surgical subtotal thyroidectomy resemble those following I^{131} therapy, indicating that there is some thyrotropic hormonal effect. It is believed that the adenomas develop in I^{131} treated thyroids as a result of thyrotropic hormonal stimulation of thyroid tissue still capable of proliferating, despite radiation injury.

The authors conclude that "until a method is found to prevent the development of these nodules, it is inadvisable to treat the younger patients with I^{131} and, if for adequate reasons, treatment with I^{131} in those under 21 years of age is required, a dose of I^{131} sufficient to destroy the thyroid gland be given and thyroid replacement therapy should be instituted."—*Charles W. Cooley, M.D.*

Buschmann, Otto, and Kaestner, Angelika. Erfahrungen mit der Radiophosphorbehandlung bei der Polycythaemia vera. (Results from treatment of polycythemia vera with radioactive phosphorus.) *Strahlentherapie*, June, 1961, *115*, 303–309. (From: Zentralröntgeninstitut und Prof. Hans Meyer-Klinik der Städt. Krankenanstalten, Bremen, Germany.)

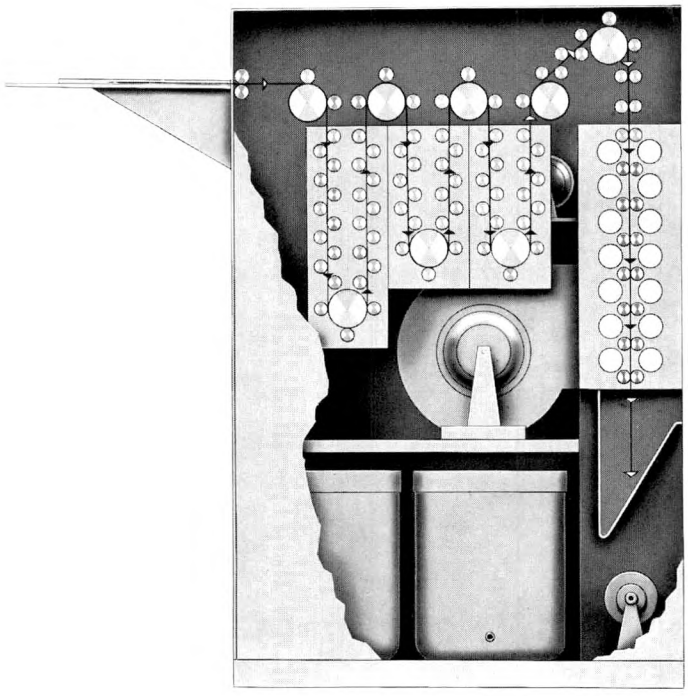
The authors review 31 patients (17 males and 14 females with an average age of 59 years) with polycythemia vera treated with radioactive phosphorus from 1950–1958. The presence of true polycythemia vera with prolonged increase in blood volume and with the associated abnormalities is the indication for treatment with P^{32}.

The authors count body weight an important gauge for dosage (they used 0.07–0.08 mc intravenously per kg. of body weight) but individualize each patient by considering also the duration and severity of the disease, any previous treatment, the patient's general condition and constitution, and the blood picture. Retreatment dosage of P^{32} depends on the therapeutic result of previous treatments. The earliest clinical effects of treatment appear in several weeks and the maximum effect at 3–4 months; therefore, retreatment should not be considered earlier than 3–4 months after the last dose of P^{32}. Results of treatment are best assessed from the blood picture: hemoglobin level, and counts of erythrocytes, leukocytes and thrombocytes.

A leukocyte count under 4,000 or a thrombocyte count under 150,000 contraindicates therapy with P^{32} which may initiate uncontrollable further drops in either count. Youth of a patient is another contraindication. The sum of all doses given should not exceed 30 mc of P^{32} for fear of irreversible bone marrow depression.

The duration of remissions averaged 15 months after treatment with P^{32}. The frequency of thromboses and emboli in patients with polycythemia vera is much decreased by treatment with P^{32}. Whether there be a leukemia-inducing effect from such treatment remains moot. Of these 31 patients, 1 died of an acute myeloblastic leukemia and 1 of chronic myelogenous leukemia. Nine other patients died: 6 of thrombotic complications, 2 of gastrointestinal bleeding, and 1 of gangrenous appendicitis.

Roentgen therapy of patients with polycythemia vera must not be forgotten; *e.g.*, when dealing with younger patients; when complications can be anticipated from therapy with P^{32}; and when retreatments approach the maximum permissible total dose for P^{32}.—*Henry G. Moehring, M.D.*



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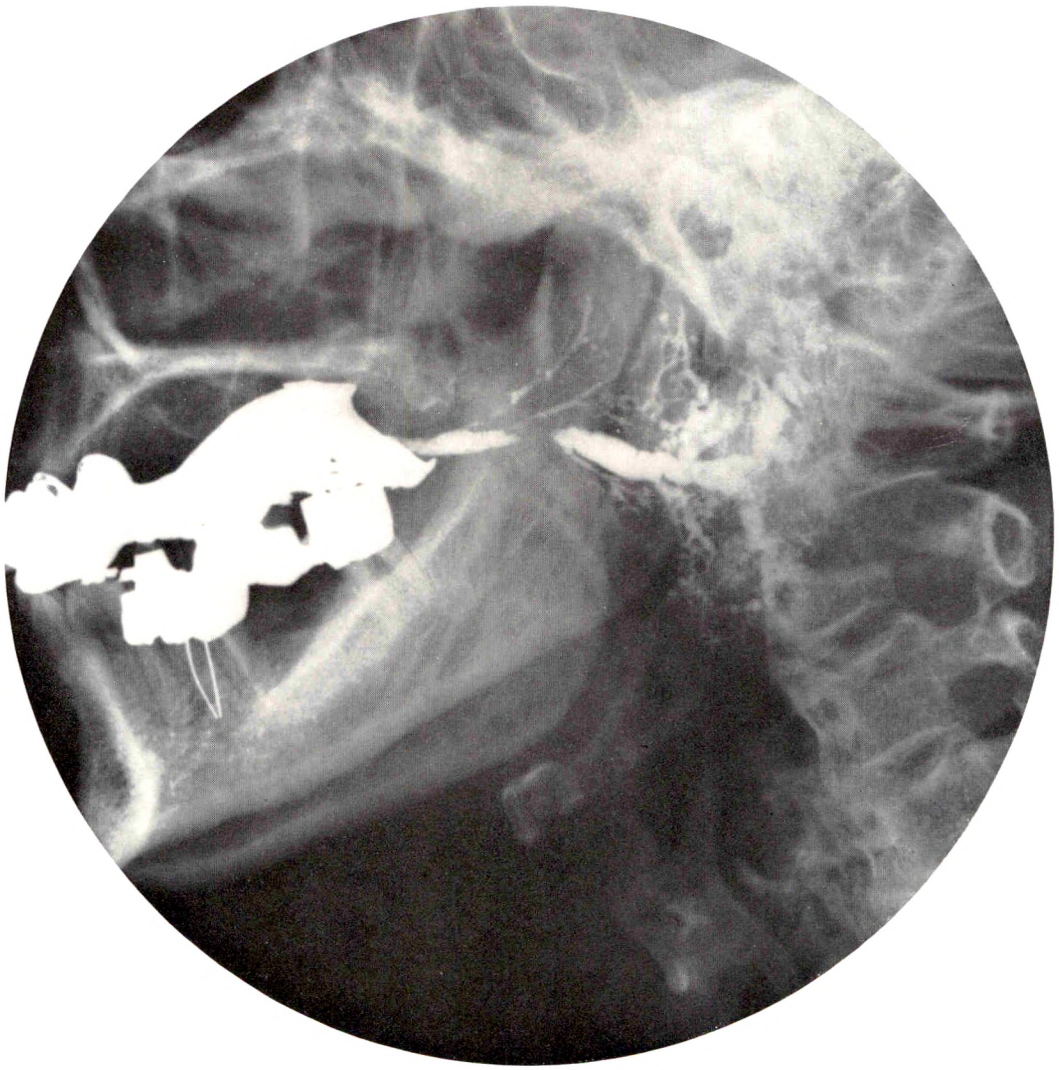
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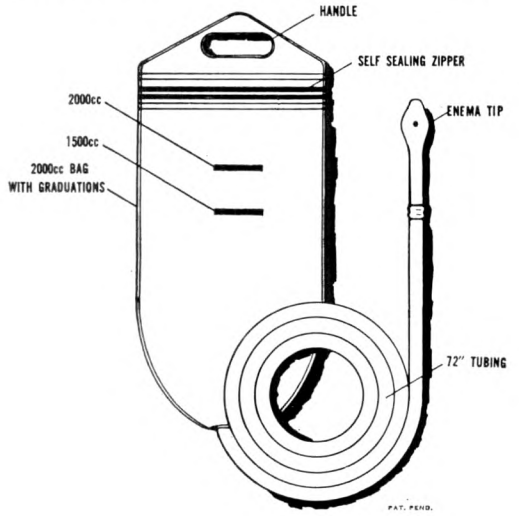
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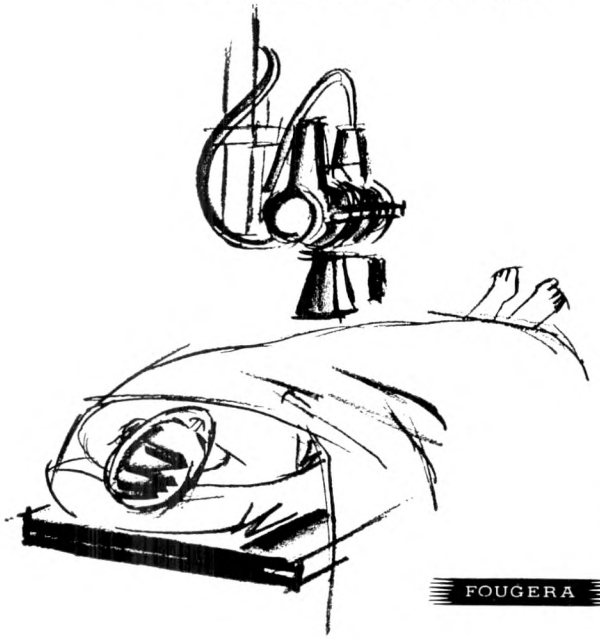
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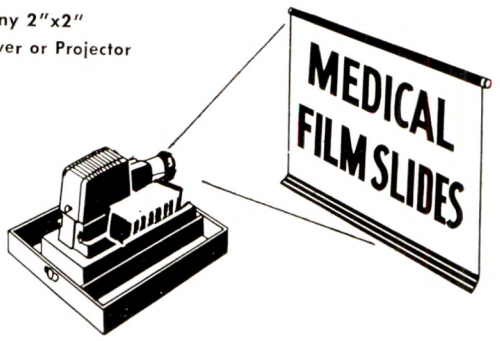


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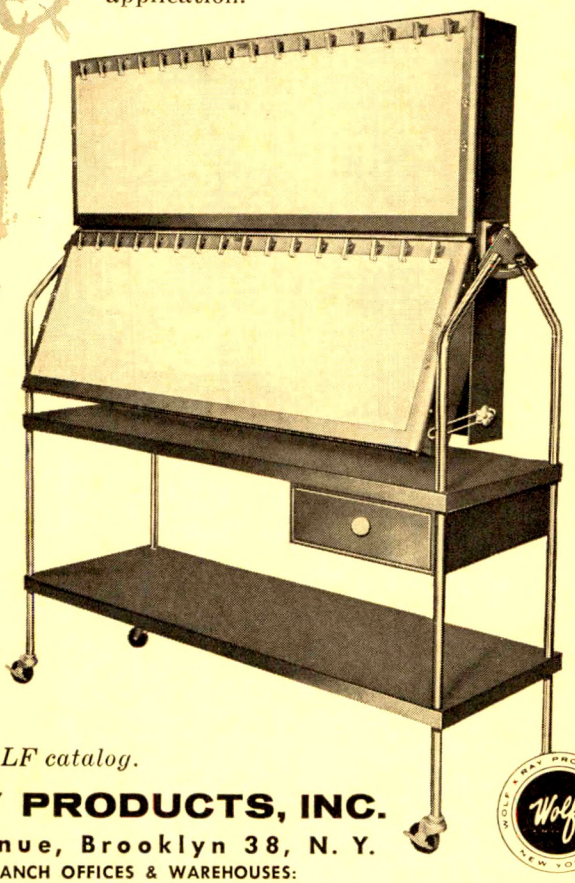
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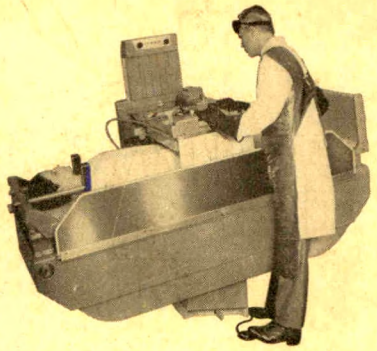
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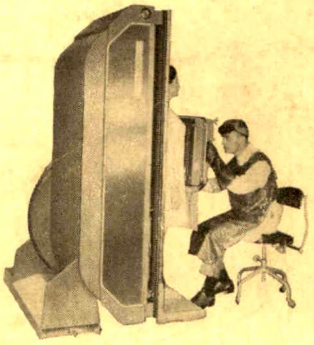
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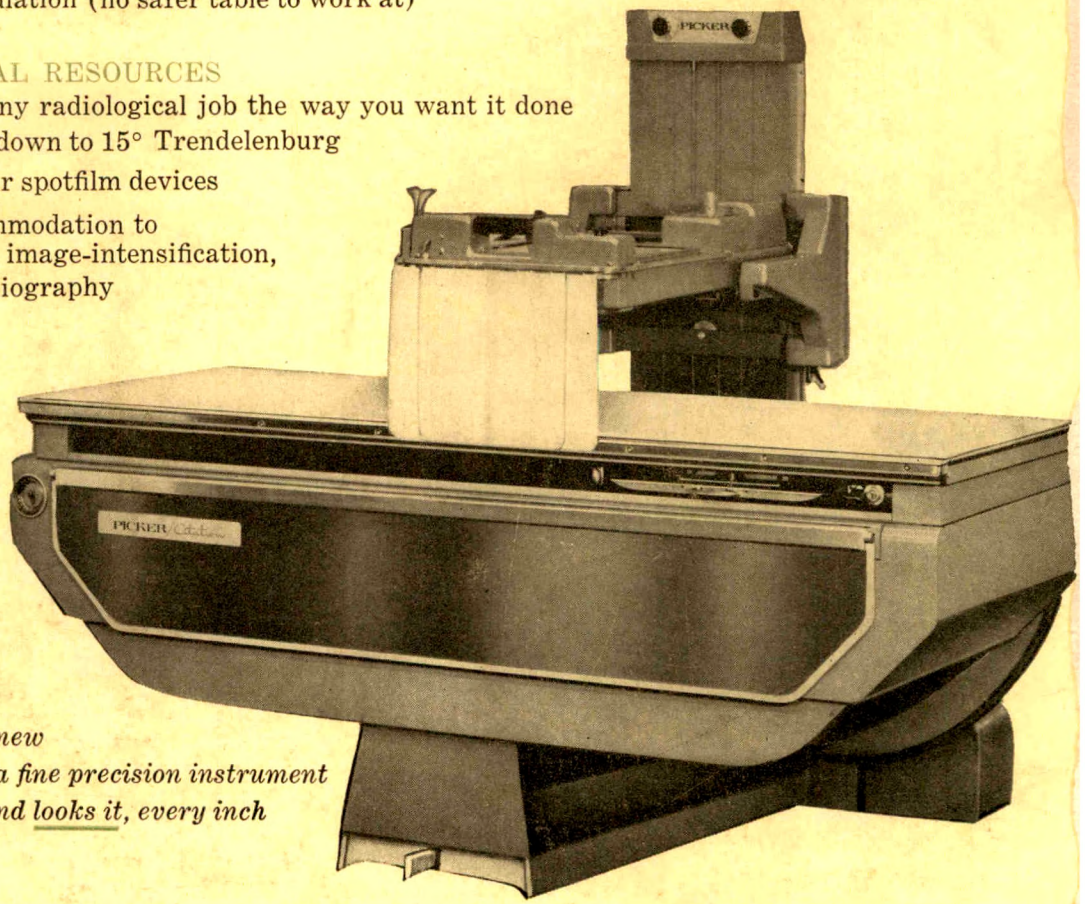
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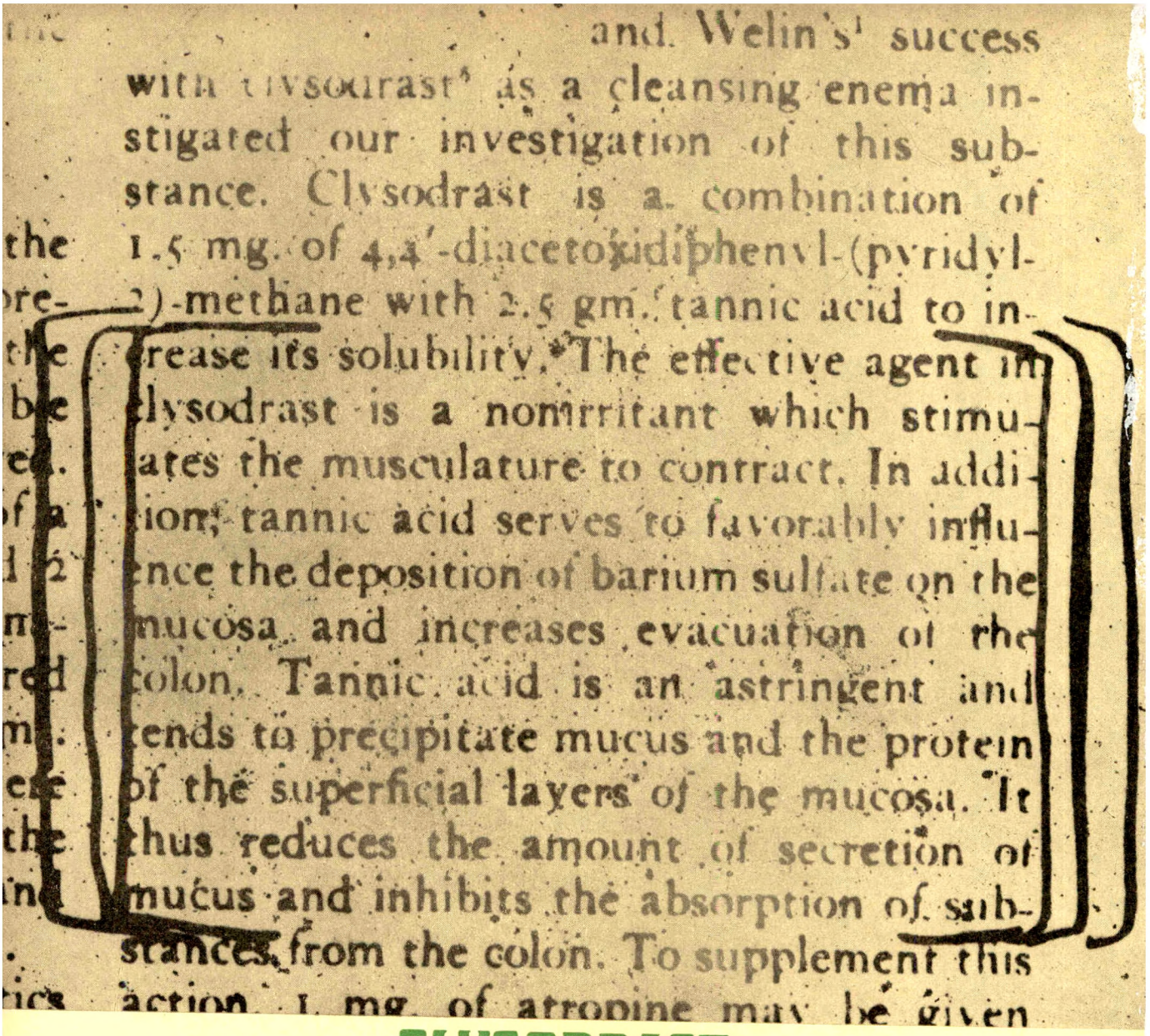
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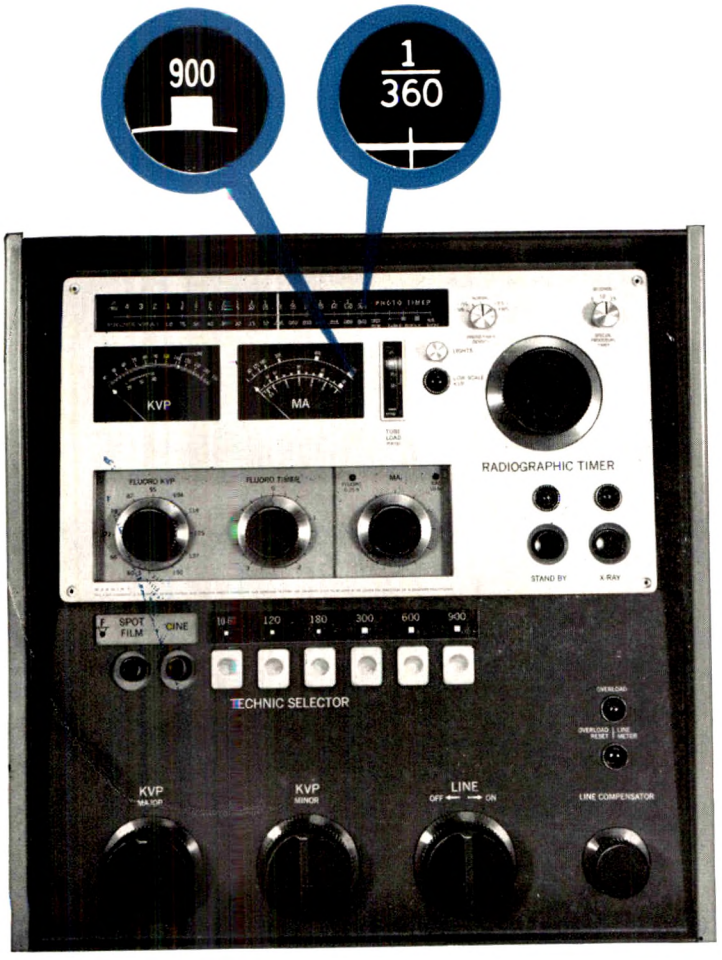
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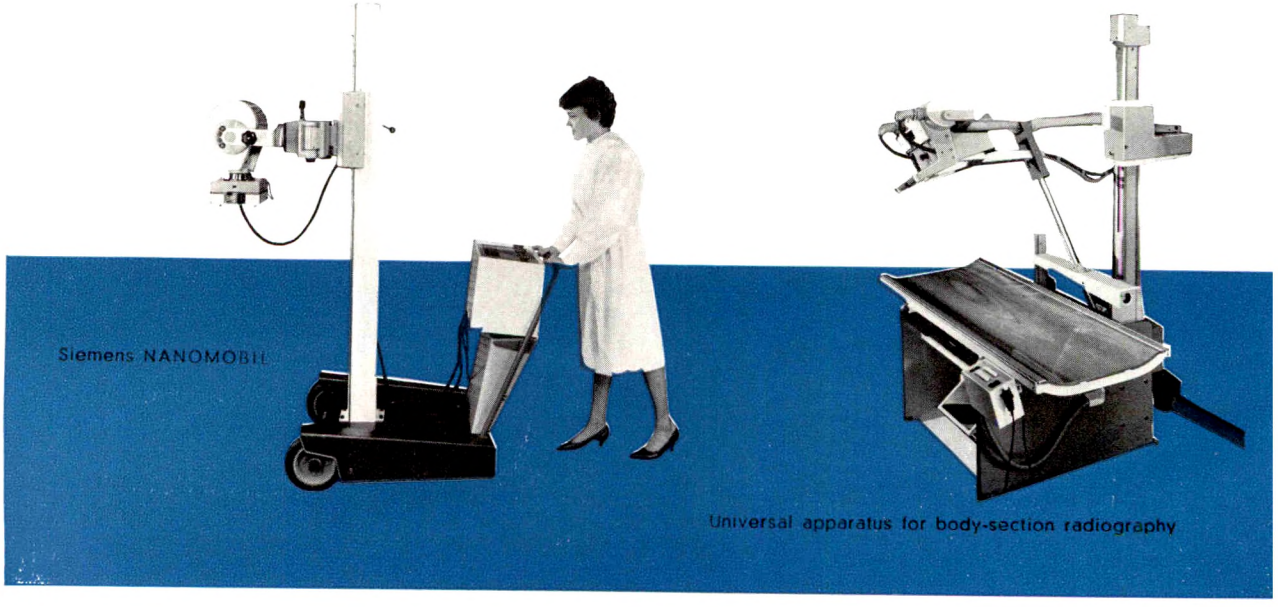
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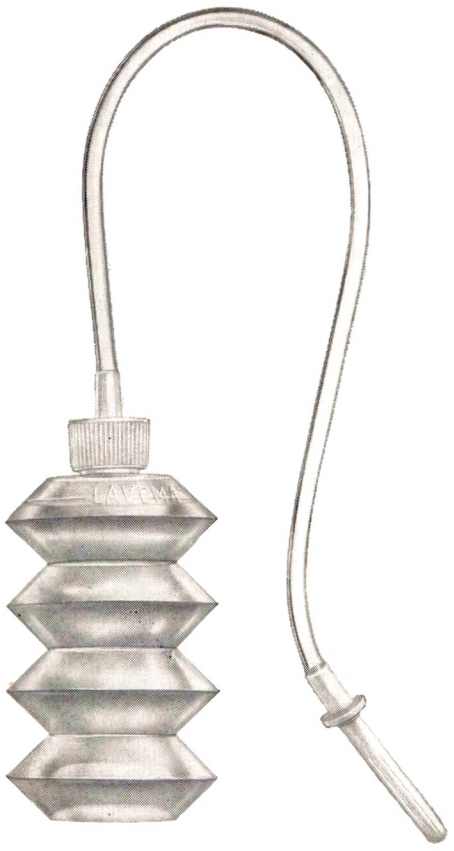


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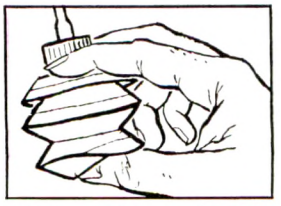
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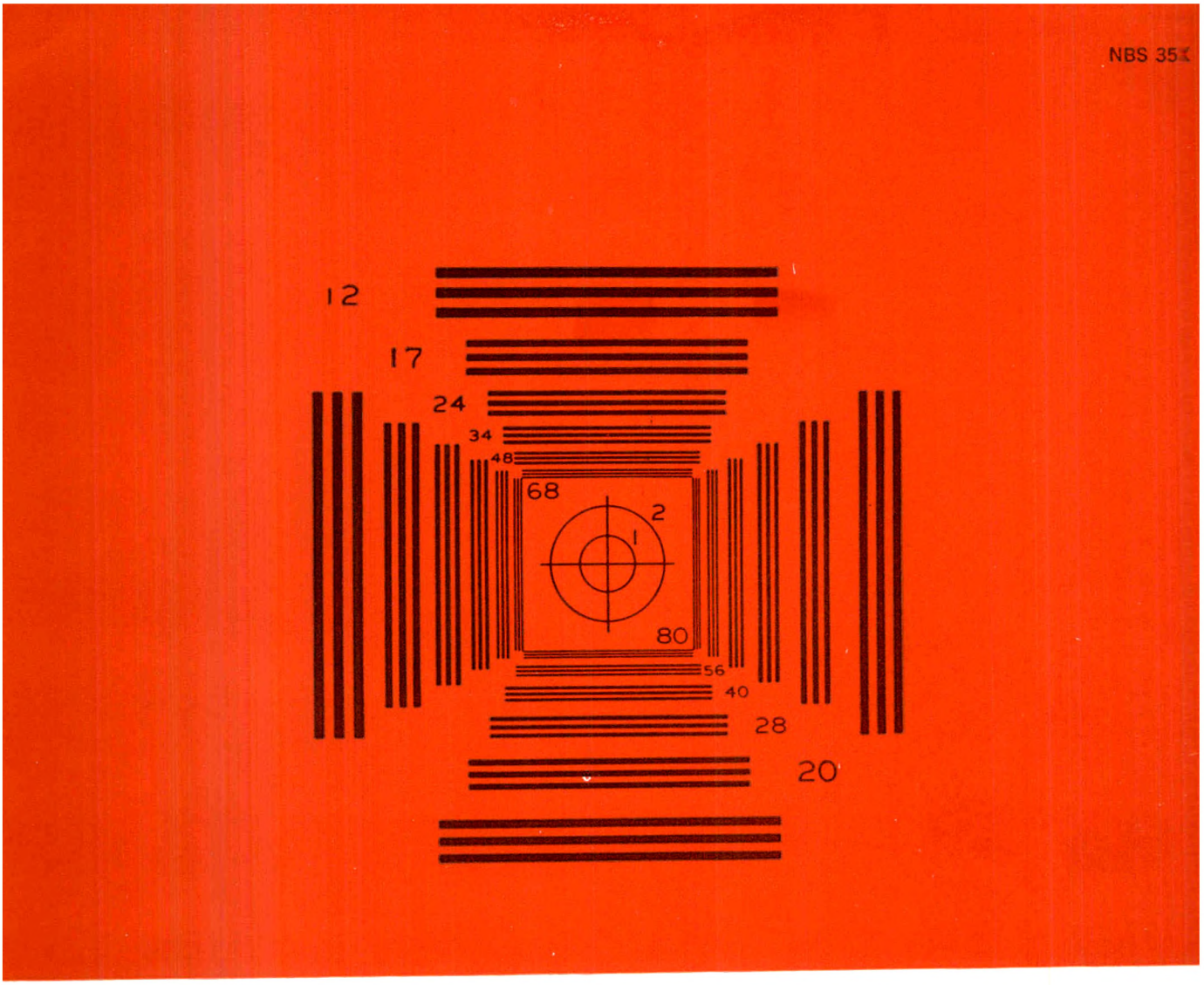
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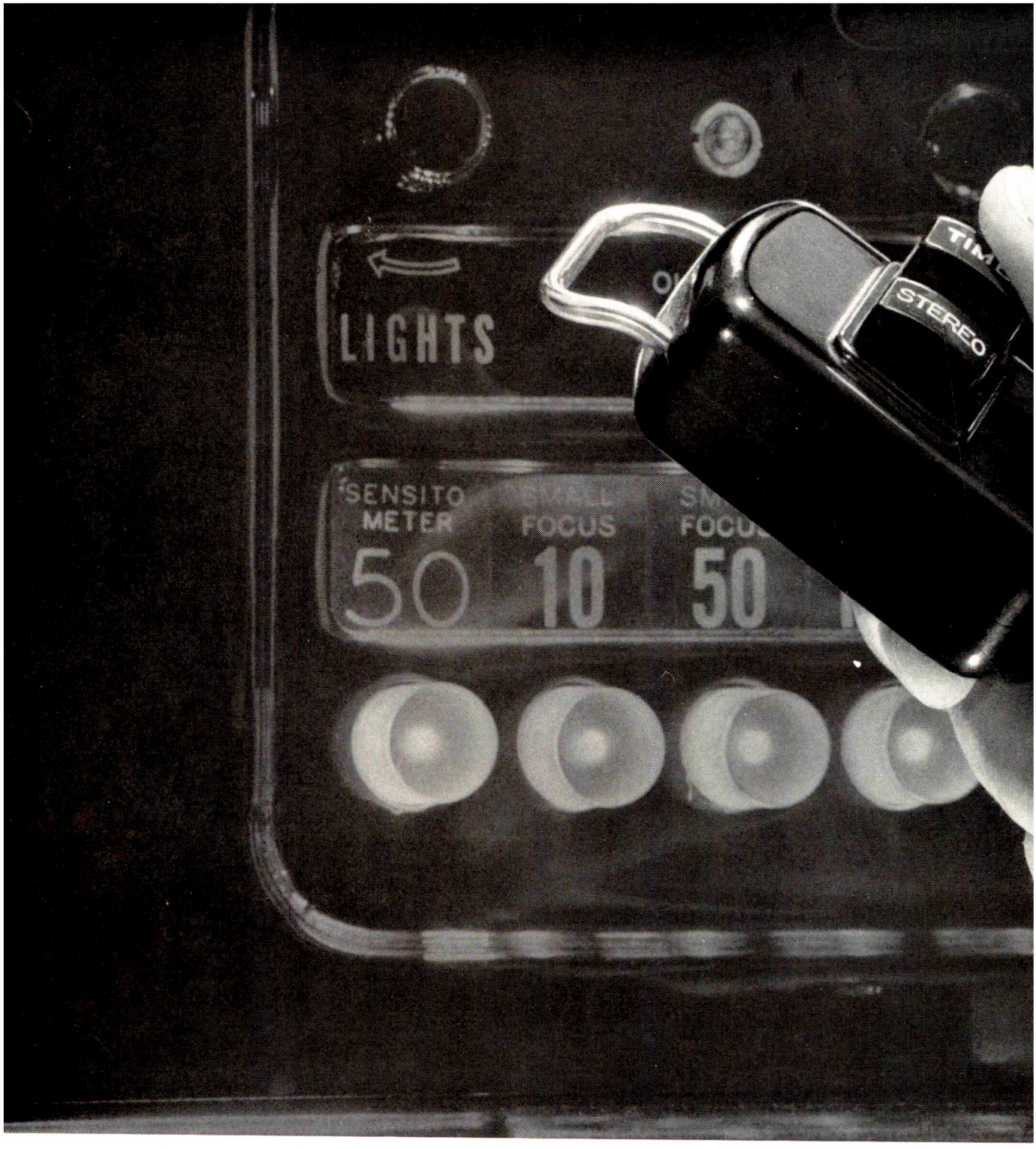
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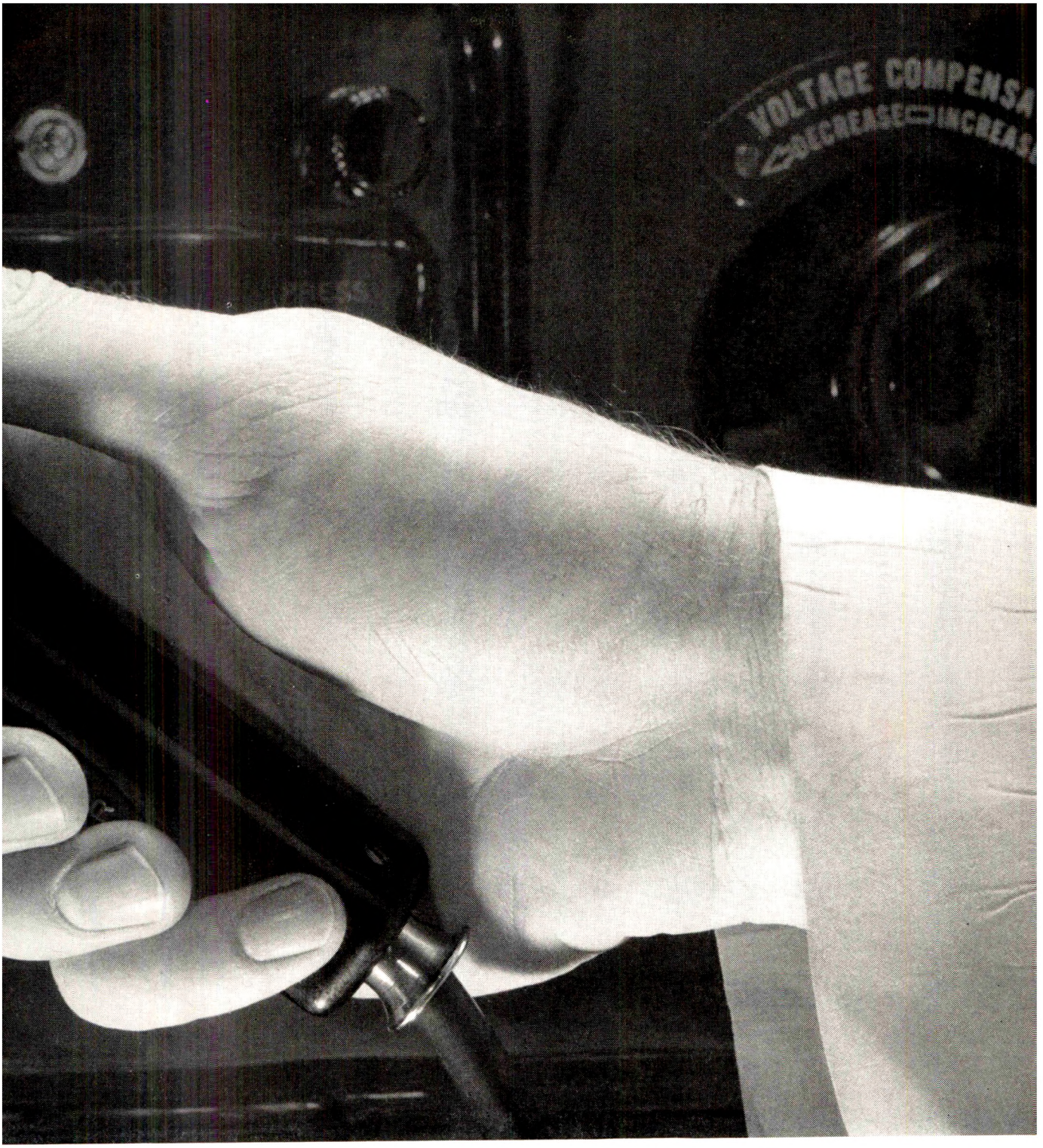
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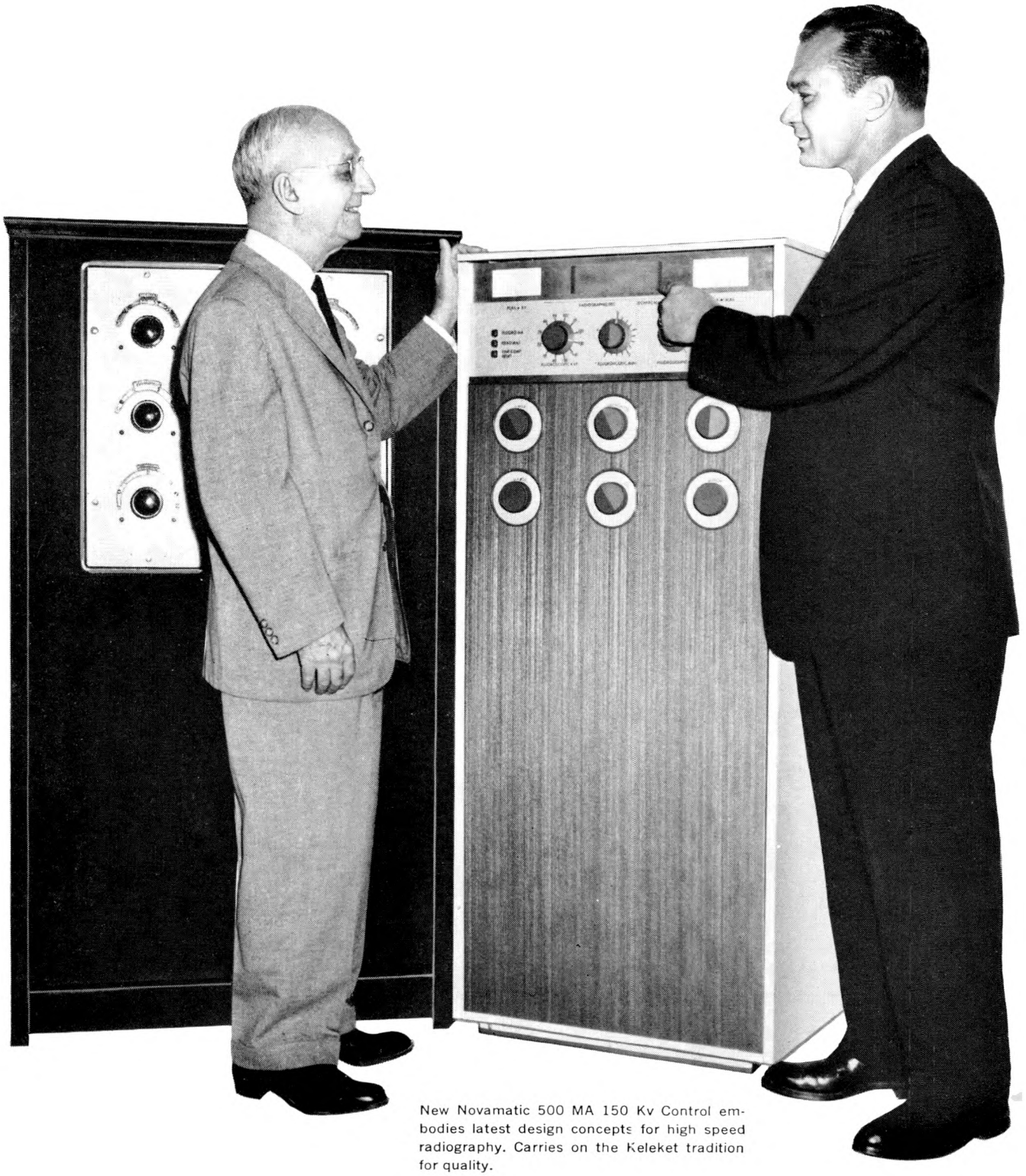
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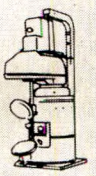
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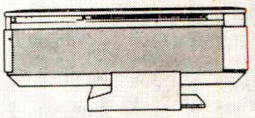
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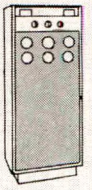
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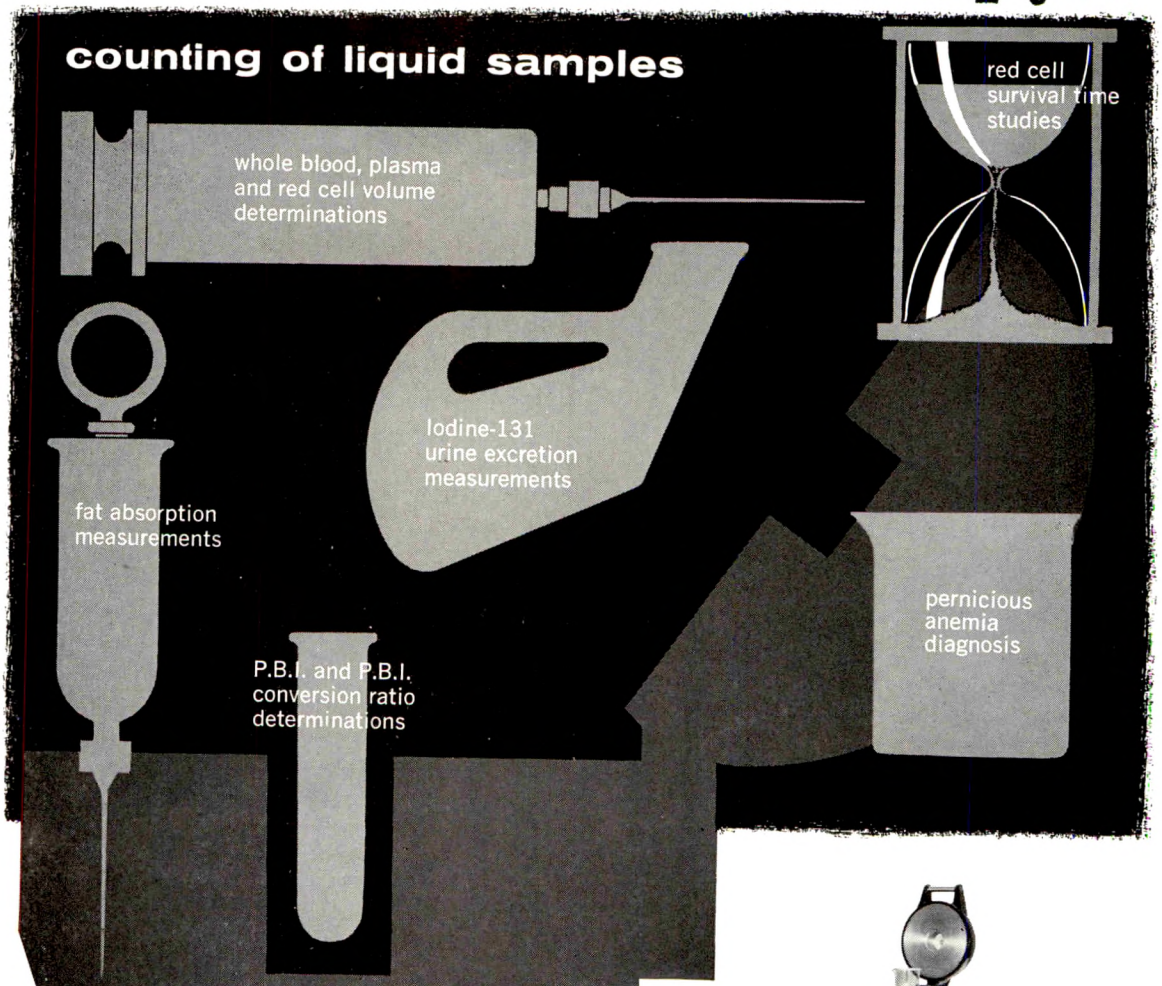


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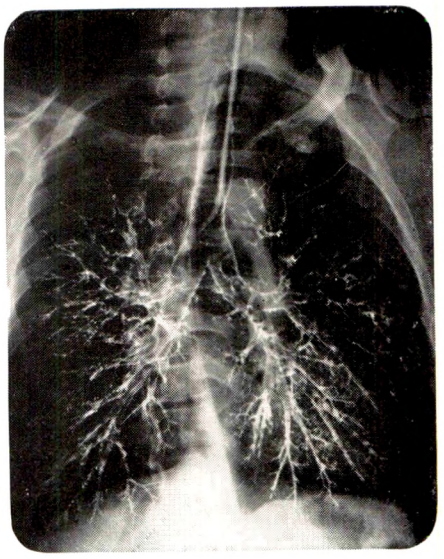


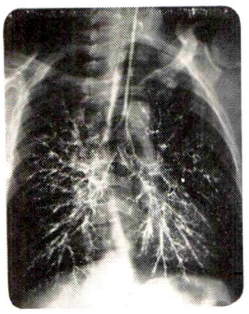
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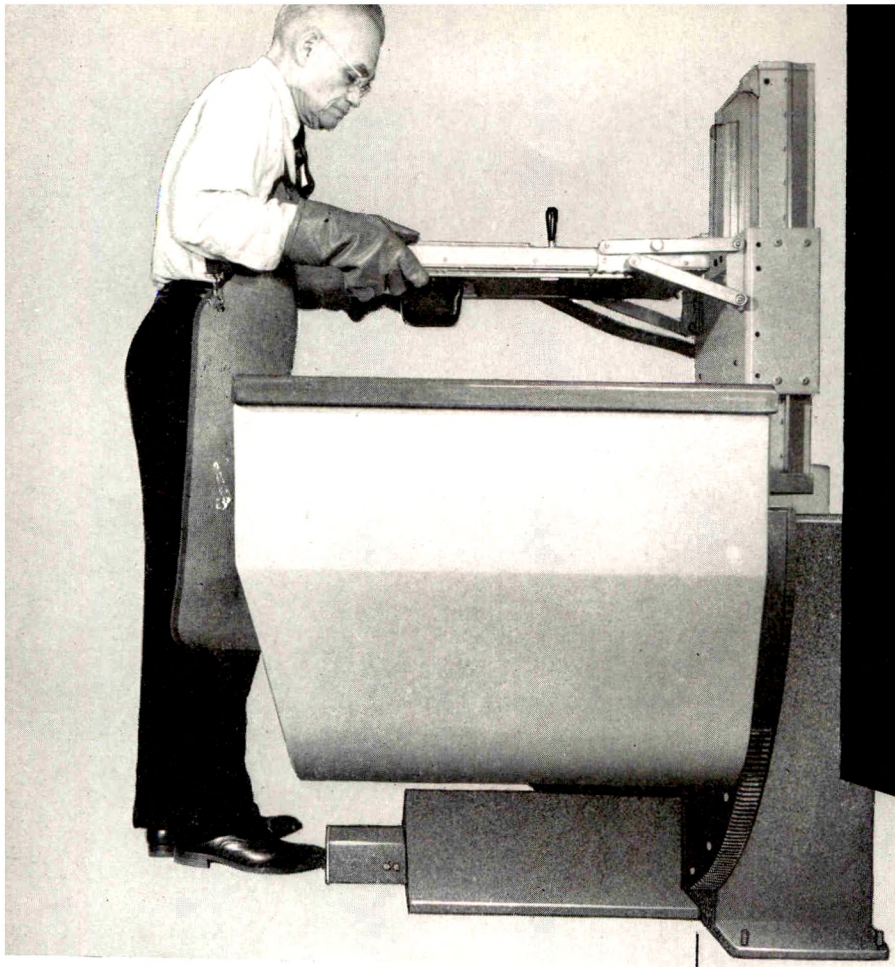
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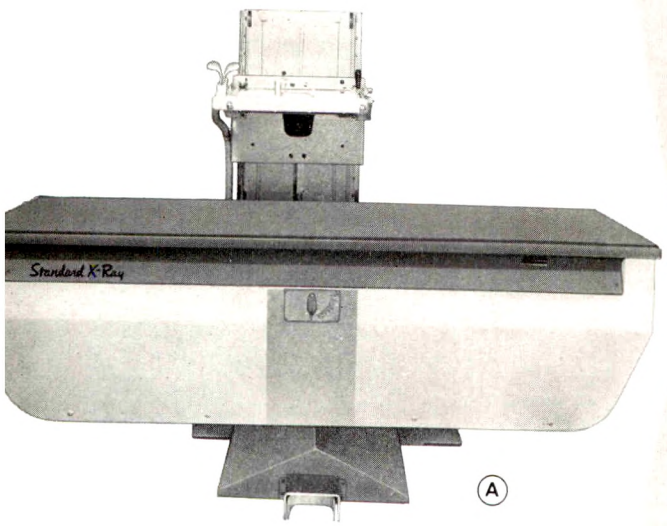
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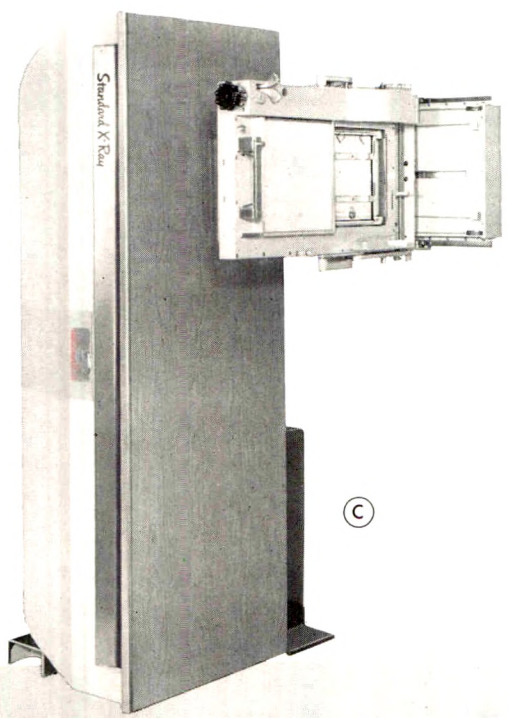
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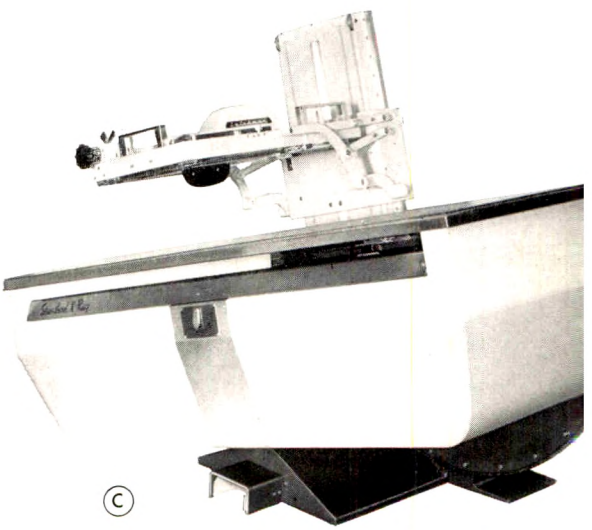
BUILT-IN PROTECTION: Adequate protection is a "must" and this table certainly has it! Completely enclosed body and automatic bucky slot cover give ample protection. A retractable radiation shield is optional equipment.

YEARS OF TROUBLE-FREE SERVICE: This is a first line, heavy duty table designed for the exacting radiologist, and built to stand up under the heaviest hospital service. Why? because it has the largest bearings of any table on the market, the best materials and the simplest mechanisms throughout. It is the finest example of Standard's 50 years of experience in the field.

TOP VALUE! This table is all new from the ground up! It is a heavy duty, quality table, that feature-for-feature provides more basic value for the price! Others may be lower priced (we make them, too), but none outshines this as a better buy!



Ⓒ



Ⓒ

Standard X-RAY COMPANY

STANDARD X-RAY CO.

IN ORAL CHOLECYSTOGRAPHY

DISTINCTLY BETTER VISUALIZATION DUE TO HIGH / RAPID ABSORPTION

The degree of visualization you may expect from an oral diagnostic agent is directly related to its absorption index, and to its selective elimination in the bile by the liver.

An ingested dose of ORABILEX is maximally and rapidly absorbed from the small intestine. Peak blood levels are attained in 1 to 2 hours, as shown by radioisotope studies.[1,2] Thus, ORABILEX delivers more opacifying iodine to the gallbladder and ducts for distinctly better visualization with minimal intestinal residue.[3-8]

ORABILEX provides a more certain diagnosis with less toxicity and fewer side effects.

1. Lasser, E. C., et al: J. Nuclear Med. 2: 79 (April) 1960. 2. Sachs, M.: Personal communication. 3. Whitehouse, W. M., and Fink, J. E., Jr.: Univ. Michigan Med. Bull. 25: 238 (July) 1959. 4. Morgan, L. A., and Parks, R. E.: Radiology 74: 436-441, 1960. 5. O'Laco, J. P., and Wilcox, W. A.: Wisconsin Med. J., 59: 249 (April) 1960. 6. Reeves, R. J.: Am. J. Roentgenol., Rad. Ther. and Nuclear Med., 83: 843 (May) 1960. 7. Farrell, T. E., and Weldon, W. V.: Southern Med. J., 54: 54 (Jan.) 1961. 8. Tatelman, M., and Steel, W. B.: J. Michigan State Med. Soc., 60: 357 (March) 1961.

ORABILEX*

THE CONTRAST MEDIUM OF CHOICE bunamiodyl sodium



*T. M. A DEVELOPMENT OF GUERBET LABORATORIES.

DUE TO HIGH/RAPID ABSORPTION

Fewer repeats... because the reliability of ORABILEX as a diagnostic medium is such that non-visualization usually indicates the presence of hepatic or biliary disease (unless disorders of the small intestine interfere with absorption). Fewer repeats mean greater economy.

DUE TO HIGH/RAPID ABSORPTION

No need for double-triple dosage... because a single 4.5 Gm. (6 capsule) dose of ORABILEX provides more opacifying material for distinctly better visualization. Should non-visualization occur and the physician wish to secure his findings, one additional 4.5 Gm. dose may be given for re-examination the following day.

DUE TO HIGH/RAPID ABSORPTION

Early cholecystography... almost complete absorption and high concentration of ORABILEX in the bile within 4 hours, as determined by radioisotope studies,[1] gives rise to the possibility of early cholecystography if needed.

Dosage: 6 capsules approximately 12 to 14 hours before examination at intervals of 2 to 5 minutes. See package insert for complete dosage information.

Contraindications: Similar to other contrast media of this class, Orabilex is contraindicated in the presence of renal insufficiency.

Supply: Envelopes of 6 capsules (0.75 grams each). Boxes of 5, 25, and 100 envelopes. Also available in hospital-size bottles of 250 capsules.



FOUGERA

A HOUSE SERVING THE RADIOLOGIST

LIPIODOL®.....multi-purpose diagnostic
ETHIODOL®.....low viscosity contrast medium
VISCIODOL®...contrast medium for bronchography
ORABILEX™.....oral cholecystographic medium
COLO-BAR™....ready-to-eat gallbladder evacuant

E. Fougera & Company, Inc. Hicksville, Long Island, N. Y.

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the Key

to reduced radiation exposure

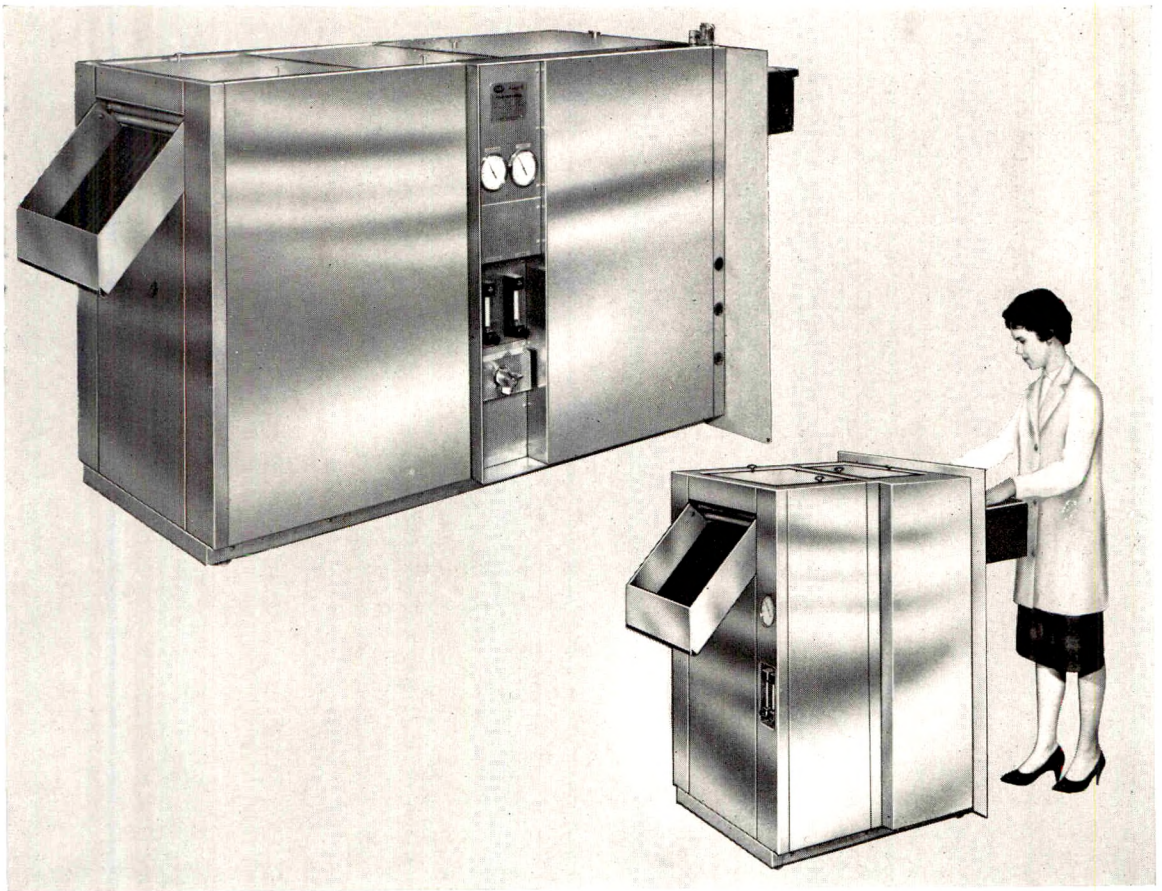
Constant research, development and perfection in Gevaert laboratories today bring you as fine and fast a film as dedicated technicians can produce. Even with a sharp reduction in exposure, your Gevaert film will enlighten your view box with a radiograph of gratifying brilliance and clarity. Seeing is believing.



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There's A Size Pakorol To Fit Your Needs, Space and Budget

Pakorol—Answer to Quality Results, Fast

Minneapolis, Minn.—Hospitals, clinics and even the smallest X-ray offices can now enjoy the benefits of automatic X-ray film processing. The larger Pakorol-X and the small Pakorol-XM each offer a fast, complete, automatic roller-type processing system at a low cost.

Fast, high quality processing and drying

The PAKOROL-X will process 120, 14″ x 17″ radiographs per hour —the PAKOROL-XM processes 65 14″ x 17″ radiographs per hour—all with uniform quality.

Space Limitations No Problem

The Pakorol-XM (miniature) is so small, neat and economical, multiple installations have often proven to be the answer to efficiency, speed and cost cutting.

Ultimate in Functional Construction

Some of the many reasons the Pakorol-X and XM drew crowds at recent conventions are: Full worm and gear driven racks, no chains; quiet roller type operation; 6½ minutes processing time (Pakorol-X) 7 minutes (Pakorol-XM) Right and Left hand models; large capacity; processes all brands of medical screen film in sizes 4″ x 4″ through 17″ x 36″ and roll film in widths of 17″ or less; No. 4 satin stainless steel finish.

Parts, Service, Training

PAKOROL EQUIPMENT is backed by established Pako Parts and Service programs. User training courses are offered regularly at the Pako factory. Let the Pako Planning Service show how the Pakorol-X and the Pakorol-XM will fit your X-ray Department. A suggested layout, tailored to your requirements and space will be prepared upon request, at no obligation.

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Please send complete data on—

☐ PAKOROL-X ☐ PAKOROL-XM

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PAKO FOR PROGRESS

Multiple-use Aristocrat-II handles special procedures, peak loads and emergencies

Aristocrat x-ray tables adapt to a remarkable variety of work situations: You'll find them in precision ordographic installations, cinefluorographic and image-intensification layouts, all sorts of specialized x-ray rooms. These *in addition* to use in literally hundreds of general diagnostic rooms!

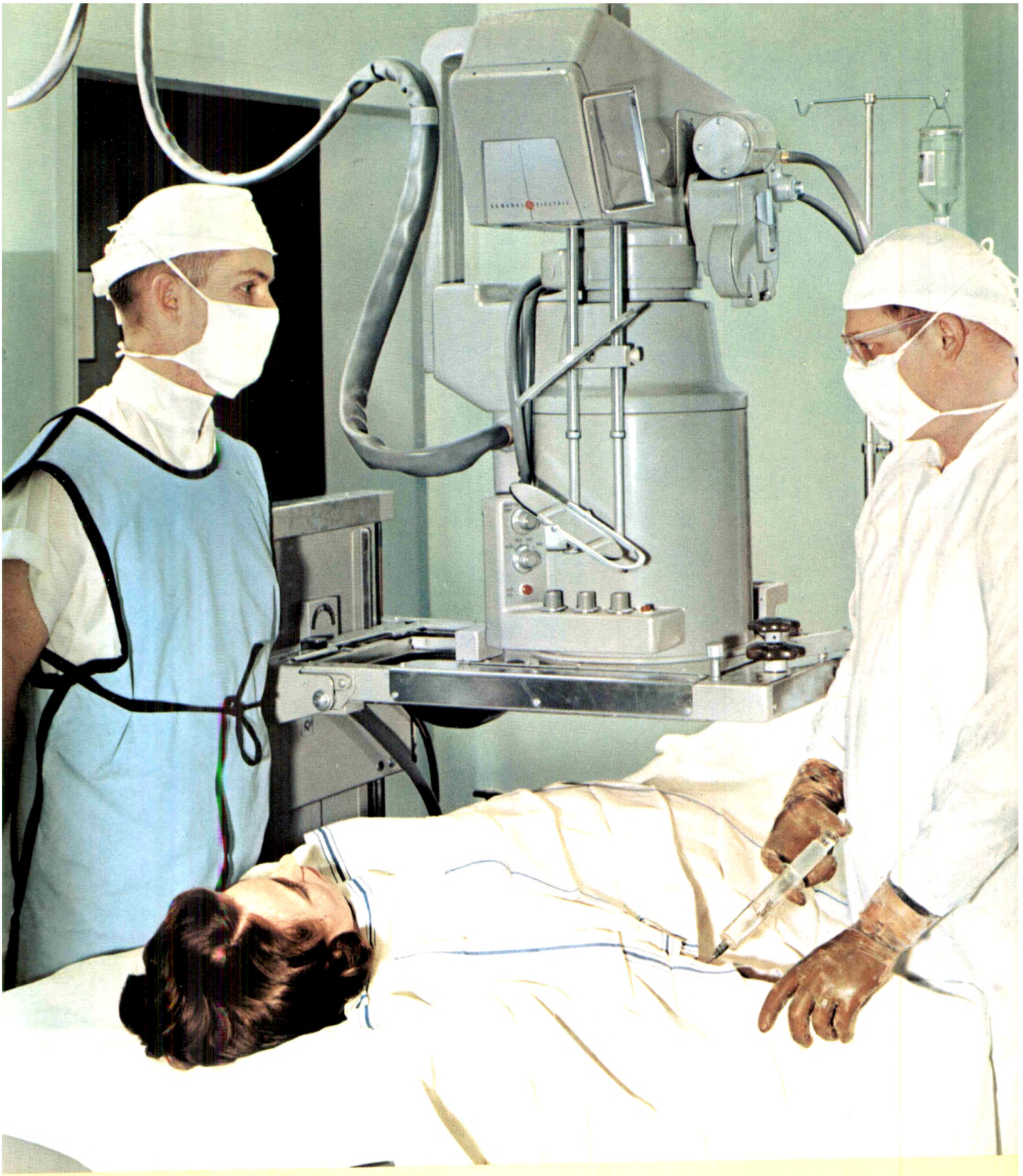
Aristocrat provides "medical center" x-ray versatility; yet keeps costs well within the desirable medium range; tailors itself to the majority of practical needs. One example of this is its 15° Trendelenburg angulation — ample for 9 out of 10 installations — and the source of substantial savings for you. Along with this there's its unusually comprehensive selection of optional components; elements that fit the Aristocrat to just about every conceivable application.

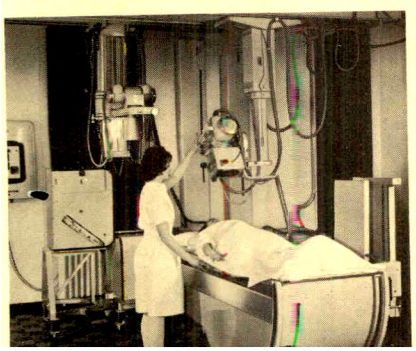
With today's trend toward greater x-ray room specialization, it's easy to see why so many Aristocrats are finding their way into advanced x-ray set-ups! Have your General Electric x-ray representative show you how an Aristocrat can help you achieve the ideal all-around balance—in economy, sophisticated performance, and lasting reliability.

Progress Is Our Most Important Product

GENERAL ELECTRIC

PRODUCTIVE PARTNERSHIP
Opposite page shows two markedly different applications of the basic G-E Aristocrat-II x-ray table (both in the same hospital).



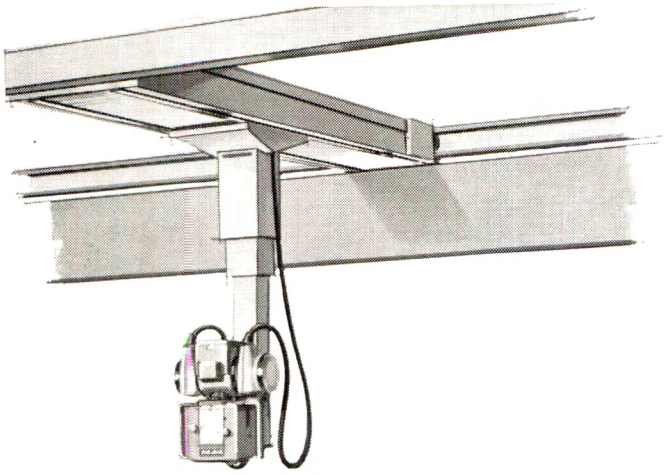


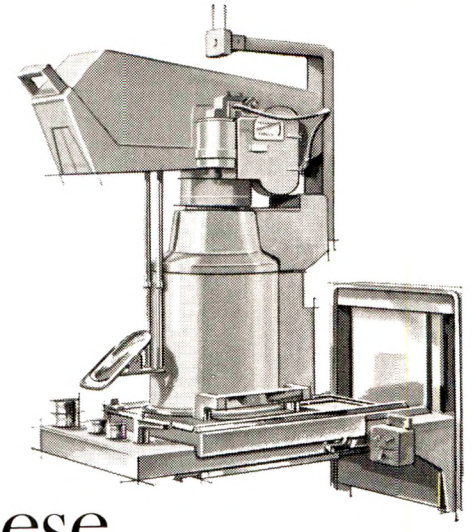
CREIGHTON UNIVERSITY SCHOOL OF MEDICINE, Omaha, Nebraska, maintains a cardio-pulmonary laboratory at local Creighton Memorial St. Joseph's Hospital. Here an Aristocrat diagnostic x-ray table, with G-E Fluoricon Image Intensifier and 16-mm cine fluorographic camera, provide an installation permitting both direct visualization and permanent recording of intricate cardiac and pulmonary studies. Staff physicians find it an invaluable aid in diagnosis because of its convenience of use and superior results.

CREIGHTON MEMORIAL ST. JOSEPH'S HOSPITAL, Omaha, Nebraska, finds multiple use for this special-procedures room in their x-ray department. The core of this installation is an Aristocrat diagnostic x-ray table and G-E Model 48-2 radiographic tube hanger. Combined with a variety of specialized equipment, these basic units can be adapted to such procedures as body-section radiography, bi-plane radiography, and vertical chest work. Cart radiography is also convenient, because of the spacious room and easy tube hanger maneuverability. (See prior page for description of **second** Aristocrat/Fluoricon installation, shown here in inset.)



LITHO IN U.S.A.



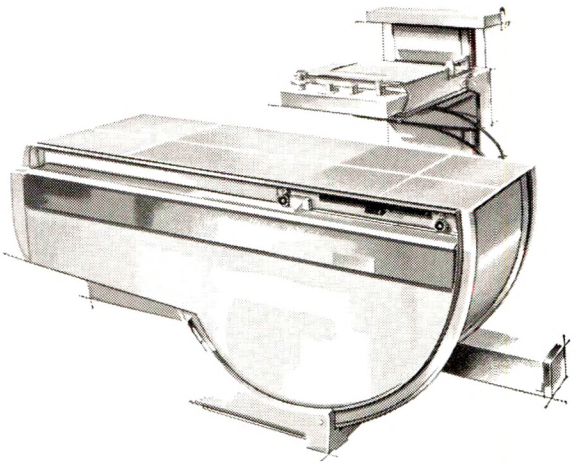


Aristocrat-II now offers all these "line leader" x-ray advantages

Probably the two most sought after attributes in x-ray equipment are *flexibility* and *stamina*. Tables must take all punishment imposed by heavy schedules. Even more important, they should assure free reign for diagnostic procedure. Aristocrat-II excels in both: offers full 105° powered angulation; automatic stop at horizontal; enclosed table, completely equipped with advanced safety features (fluoroscopic shutter limiting; self-closing Bucky-slot; etc.). Super-Speed or Par-Speed Reciprocmatic Bucky; accepts interchangeable grids, including high-kv types. Choice of G-E automatic spot-film devices.

• A very popular Aristocrat elective is the G-E Model 48-1 tube hanger; frees x-ray positioning from the frustrations of ordinary systems. Also lets you deploy auxiliary radiographic equipment right where there's ample room to work. Counterbalanced "reflexomatic" tube positioning. Convenient "disappearing" overhead cables.

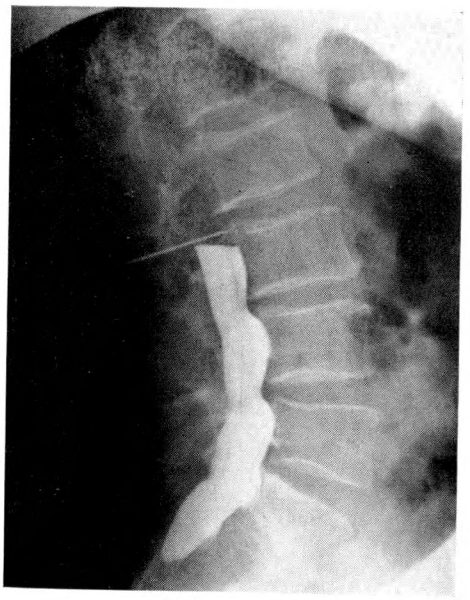
• Focus of keenest interest for radiologists is the brilliant G-E Fluoricon image intensifier that permits direct fluoroscopic viewing in normal ambient light. This too is available for use with the Aristocrat-II, to further extend its versatility and help you obtain the advanced facilities of a completely-equipped diagnostic installation.

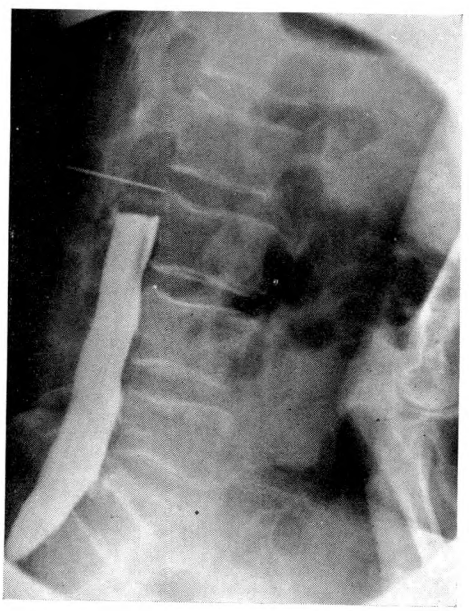


For complete details on the G-E Aristocrat-II x-ray table and options, contact your General Electric x-ray representative; or write to X-Ray Department, General Electric Company, Milwaukee 1, Wisconsin, Room A-81.

Progress Is Our Most Important Product

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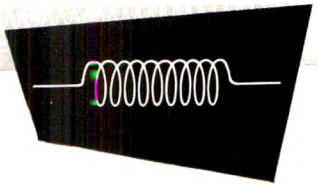
Dynamic Myelography

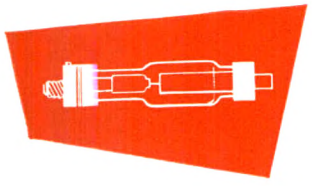
These two radiographs of the same patient demonstrate the bulging of the annulus fibrosus during hyperextension and flexion, respectively, of the vertebral column. 30 cc of ***Pantopaque*** contrast medium was used. Note how this technique permits visualization of the posterior surface of the vertebral canal.

"PANTOPAQUE" *is the registered trademark under which all leading x-ray dealers supply the compound ethyl iodophenylundecylate, which is synthesized by the Research Laboratories of Eastman Kodak Company and prepared as the myelographic contrast medium Iophendylate Injection, U.S.P., by Lafayette Pharmacal Inc. The trademark serves to indicate to the radiologist continuity of experience in the manufacture of this medium.*

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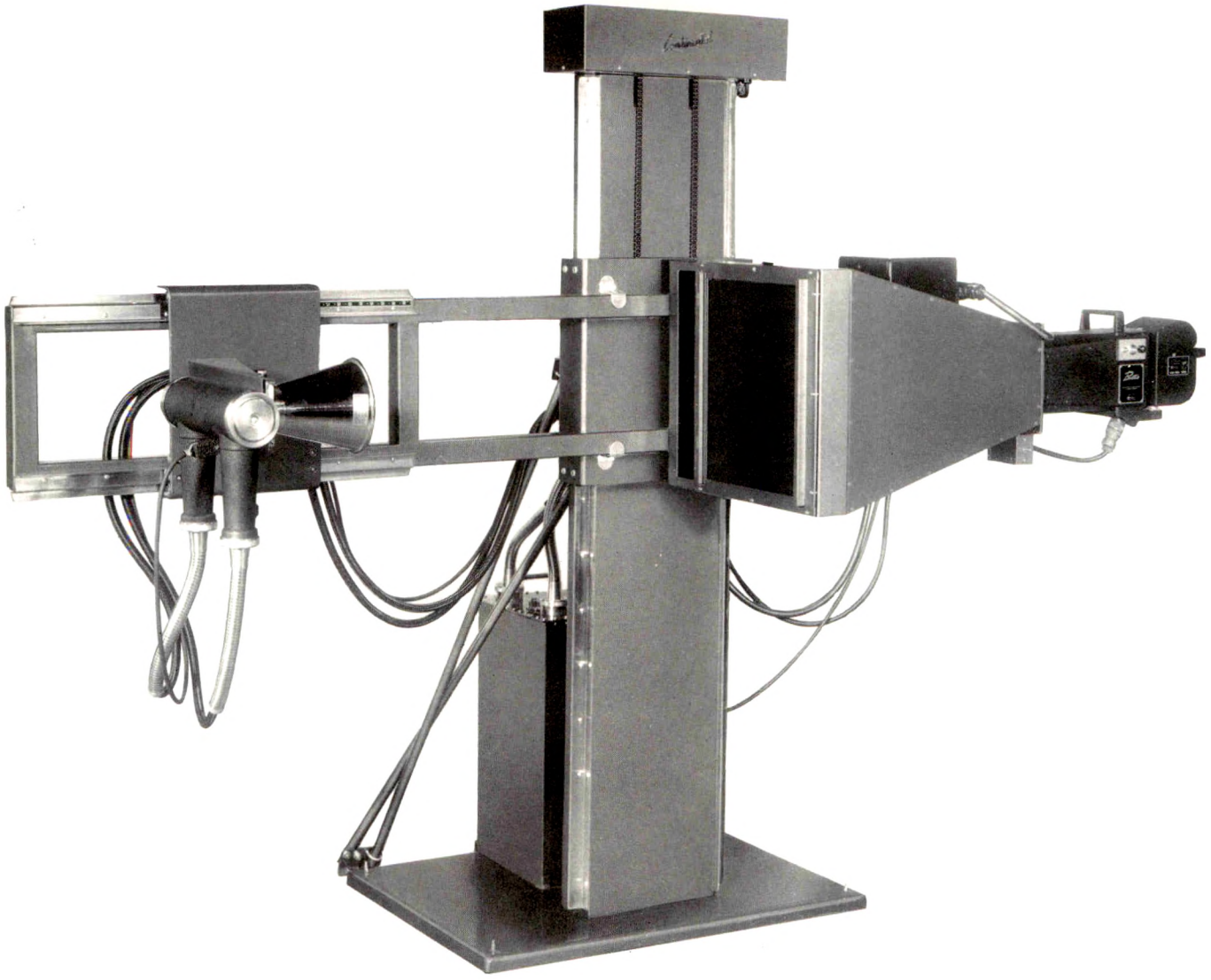
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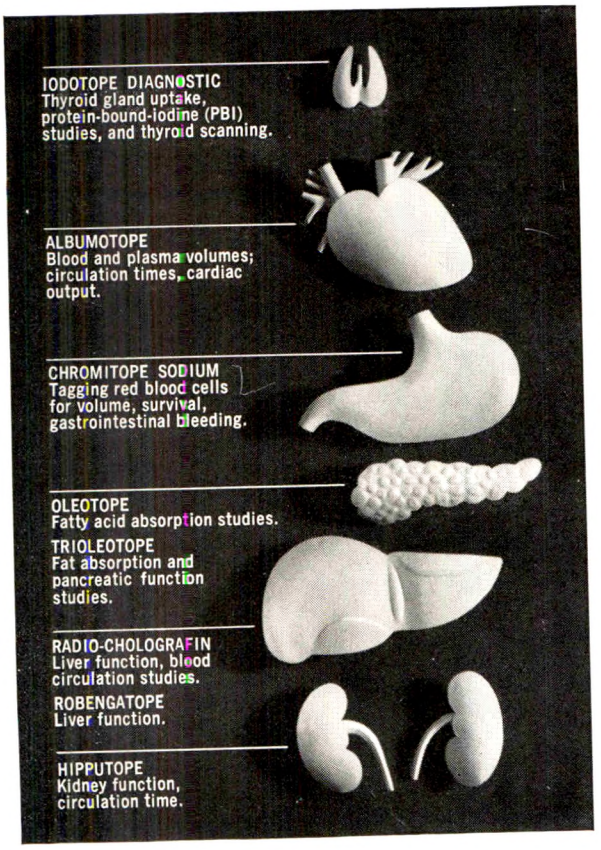
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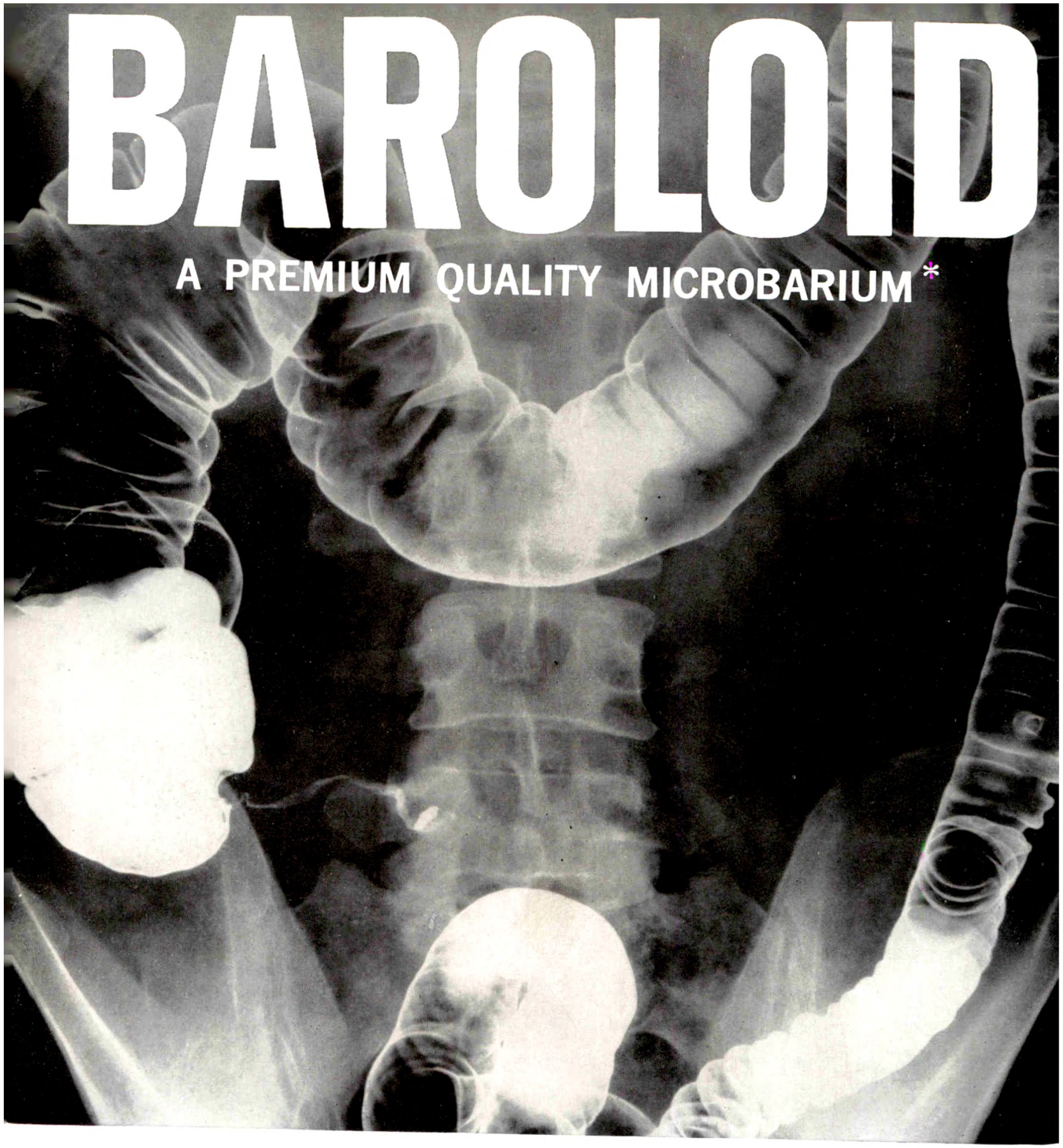
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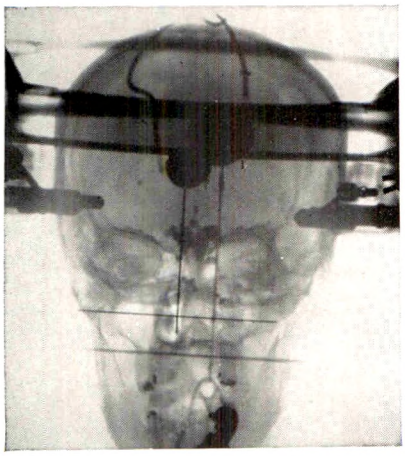
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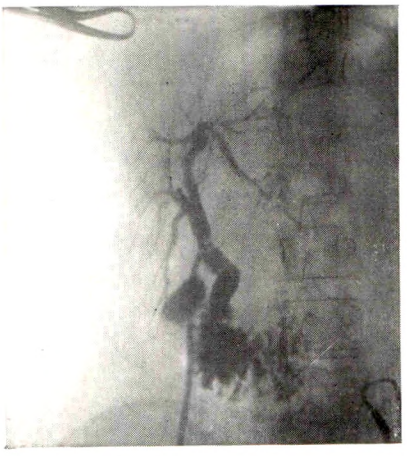
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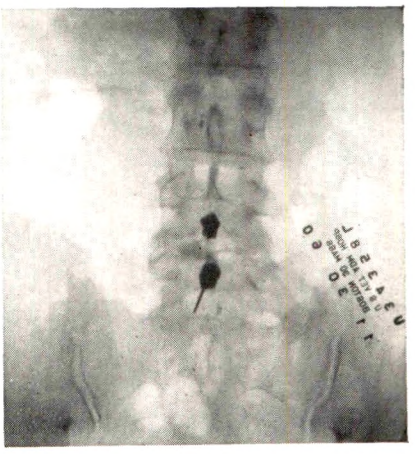
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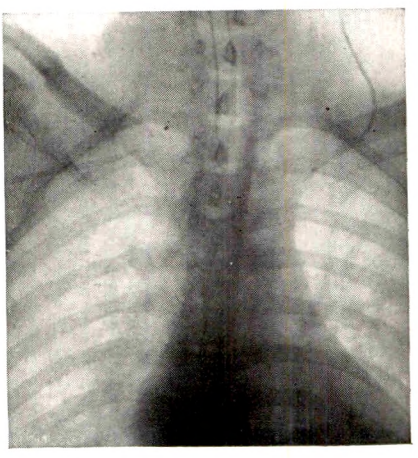
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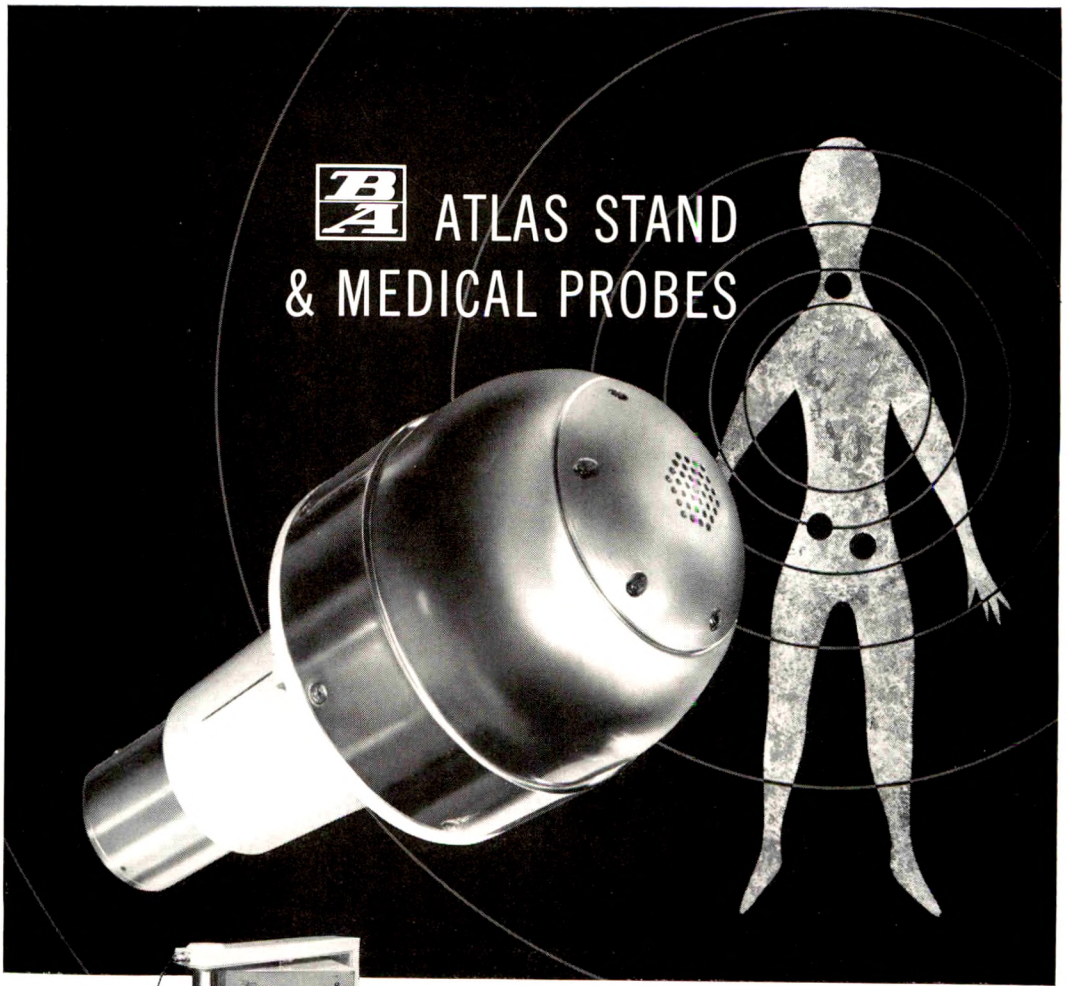
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plenty of **latitude,** too — There's ample exposure leeway in the new Polaroid 3000X: no need for hair-line technique factors.

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The Atlas Stand is notable for smooth, effortless adjustments in any plane. Rugged and massive, it handles with ease and safety the heaviest combinations of probes, collimators, and instruments. The easily swivelled instrument cabinet allows viewing from any angle.

The Atlas Stand is designed for, and is used in conjunction with, the new line of Baird-Atomic Probes.

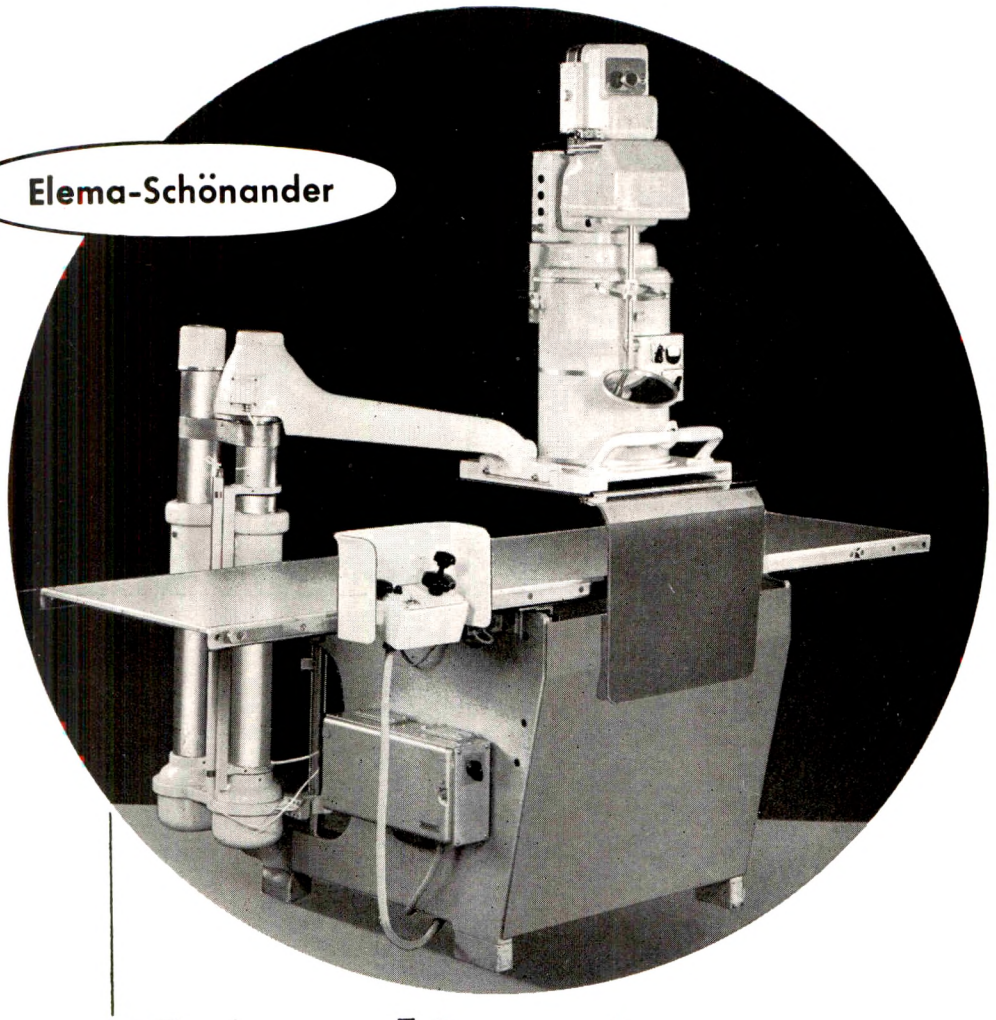
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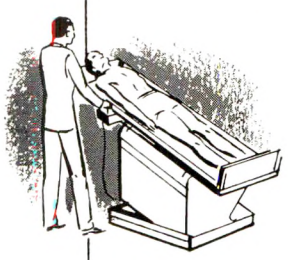
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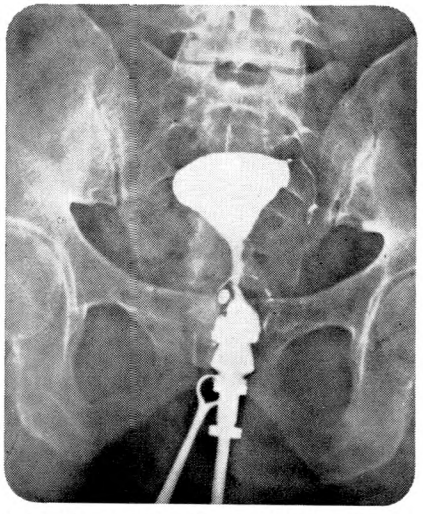
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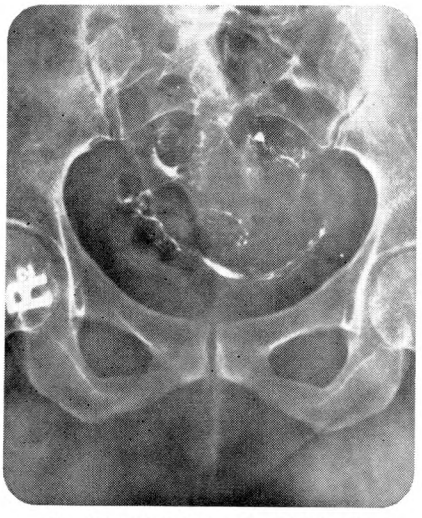
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By

ERVIN EPSTEIN, M.D.
Associate Clinical Professor of Dermatology
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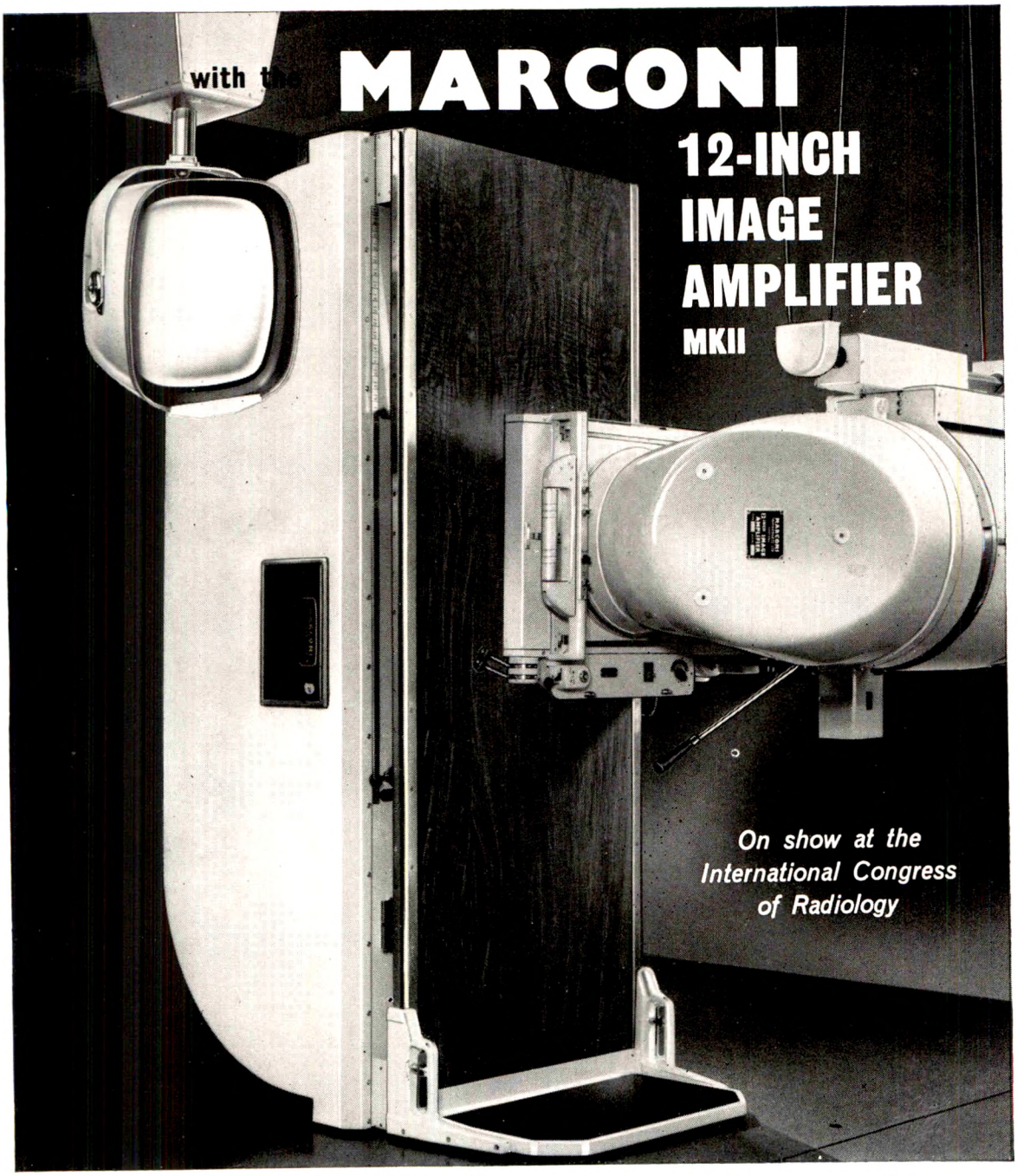
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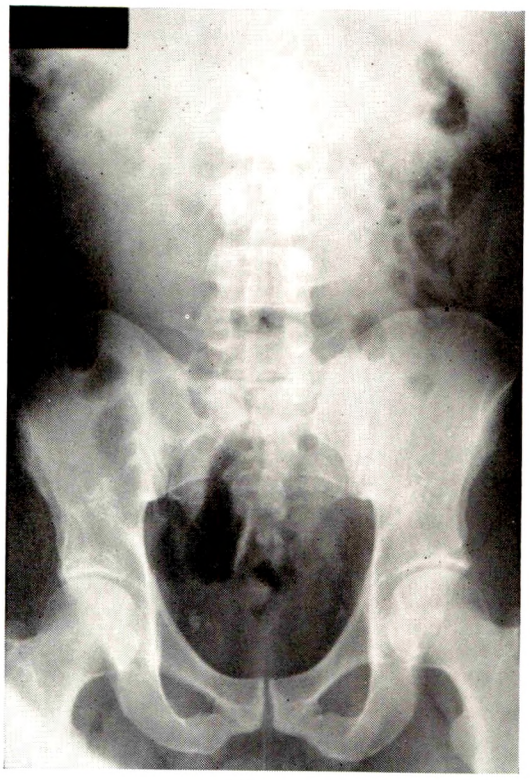
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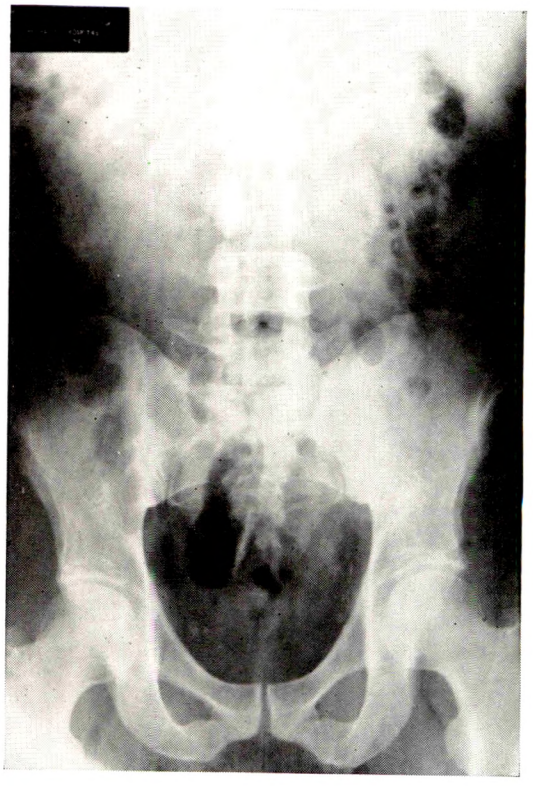
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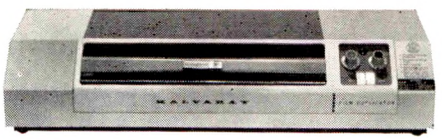


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VOL. 88 AUGUST, 1962 No. 2

TEN YEAR EXPERIENCE WITH LOW MEGAVOLT ELECTRON THERAPY*

By MAGNUS I. SMEDAL, M.D.,† DAVID O. JOHNSTON, M.D.,† FERDINAND A. SALZMAN, M.D.,† JOHN G. TRUMP, D.Sc.,‡ *and* KENNETH A. WRIGHT, M.S.‡
BOSTON AND CAMBRIDGE, MASSACHUSETTS

DURING the 10 year period, 1951 through 1960, 522 patients have been treated with low megavolt electrons in the cooperative program of the Massachusetts Institute of Technology and the Lahey Clinic. This project was initiated by the need for an alternative means of continuing radiation therapy in a 76 year old patient with recurrent total cutaneous involvement by lymphoma cutis (mycosis fungoides variant). The radiation tolerance dose had already been exceeded from repeated courses of spot and spray irradiation with superficial and medium kilovolt roentgen ray apparatus.

The advantages of applying the unique properties of megavolt electrons to the treatment of superficial diseases had been suggested by Trump, Van de Graaff and Cloud[6] in 1940. In 1951 certain modifications to provide for the emergence through a wide slit of a downward-directed beam of electrons were added to a low megavolt Van de Graaff electron accelerator already available at the High Voltage Research Laboratory. The resultant radiation therapy source then met 3 principal clinical requirements: (1) controllable voltage to adjust the electron penetration to the clinically recognizable pathologic thicknesses within the skin, (2) accurate application of the desired electron dose over large areas, and (3) low accompanying roentgen ray background.

PHYSICAL ASPECTS

Previous publications from our laboratory have emphasized the desirability of monoenergetic and normally-incident electrons for the therapy of superficial disease.[3,5,7,8] Some of the physical aspects of such radiation are now summarized.

SOURCE DESCRIPTION

The source of electrons is a constant potential electrostatic generator of the

* From the †Department of Radiology, The Lahey Clinic, Boston, Massachusetts, and ‡High Voltage Research Laboratory, Department of Electrical Engineering, Massachusetts Institute of Technology, Cambridge, Massachusetts.

Presented at the Sixty-second Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 26–29, 1961.

The authors gratefully acknowledge the support of this investigation during the past ten years by the Damon Runyon Memorial Fund for Cancer Research through their grant DRMF-204 to the Massachusetts Institute of Technology.

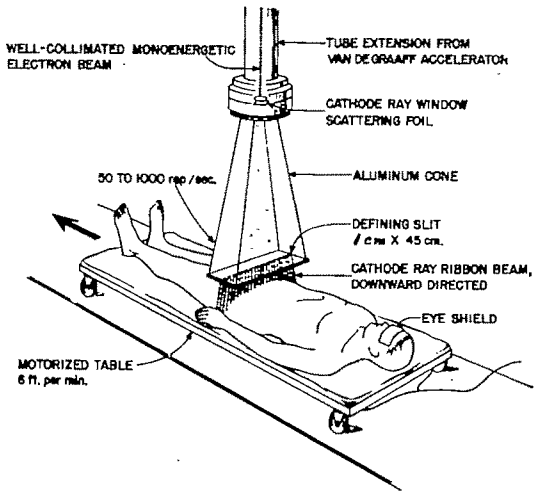


FIG. 1. Electron cone in position, with patient on motorized table. (Reproduced with permission from *Radiology*.)

Van de Graaff type, insulated in compressed gas. The electron beam emerges into air from the evacuated accelerator tube through an aluminum window of 3 mil thickness (Fig. 1), and is further scattered by an additional 15 mil of aluminum. The scattered beam is confined to an aluminum cone, the bottom of which is closed except for a slit 45 cm. long and 1 cm. wide. Interaction between the monoenergetic electron beam and the aluminum cone gives rise to the major part of accompanying radiation. To reduce the roentgen ray background, masonite which has a lower atomic number than aluminum is now used to line the inner aspect of the distal part of the cone and the slit width has been doubled to 1 cm. These changes made a four-fold reduction in the roentgen ray dose, which at present is about 10 r for an electron treatment of 600 rad to the entire body surface. More recent modifications of the cone have been studied which further reduce the roentgen ray component and diminish the electron energy losses in transit to the patient.

The electron dose is monitored and measured by continuous reading of the total beam current collected by the cone. The ratio of delivered dose to total beam current is a function of voltage; calibrations are available for the electron energy range from 1 to 4 mev.

All treatments, whether of single spots, small fields or full surface areas, are administered during transit of the patient beneath the treatment cone on a motorized couch traveling at a rate of 6 feet per minute.

CLINICAL DOSIMETRY

Typical distribution curves of dose in depth are presented in Figure 2 for electrons accelerated by potentials of 1.5, 2.5 and 3.5 mev. These curves have been measured with the same cone and moving couch technique used in treating patients. We are cognizant that both the absolute dose and the typical distribution in depth are affected somewhat by the distance between the defining cone and the patient as well as by the position along the slit. The treatment dose specified throughout this paper is the entry dose in rad. This entry dose has been used in this discussion to represent the minimum tissue dose in the estimated region of the lesion. The voltage by which the electrons are accelerated is selected to deliver this entry dose to the clinically estimated depth of the lesion. When the lesion is considered to be beyond the range at which the dose has diminished to the entry level, an appropriate increase in entry dose is made. Two mev. roentgen rays have been used for local treatment of some lesions too thick for the electron energies available.

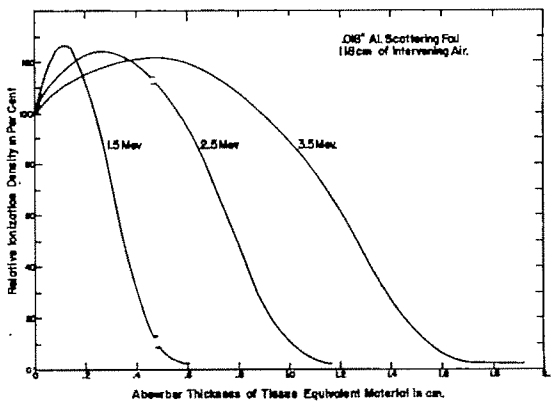


FIG. 2. Distribution of dose in depth for large fields with electrons accelerated by potentials of 1.5, 2.5 and 3.5 mv.

Because of their distribution in depth, low megavolt electrons produce a several fold lower volume dose as compared with soft roentgen rays. This is an important advantage in the treatment of extensive superficial disease, since systemic effects appear to be completely avoided.[1]

CLINICAL ASPECTS

Our interest in electron therapy evolved from the need for total body surface irradiation required for lymphoma cutis and then extended to other malignant and benign disease of the skin (Table I). The ability to localize the delivered dose to the desired depth makes low megavolt electron therapy uniquely suitable for rapid large field treatment. Small field involvement can also be advantageously treated and later receive considerable attention. The major interest, however, has been in the wide field irradiation of lymphoma cutis, Kaposi's disease, skin implants of breast carcinoma and generalized dermatoses. Primary skin cancer, keloids and isolated cases of other diseases round out the listing.

MALIGNANT DISEASE

LYMPHOMA CUTIS

The essential features of this uncommon and usually fatal disease have been discussed in previous reports in conjunction with our dermatologic associate.[4,5] Special attention is placed on mycosis fungoides which accounts for the majority of patients (220) in this group (Table I). A detailed account and analysis are beyond the scope of this report and are planned for the near future. The 3 stages of mycosis fungoides, the erythematous, the infiltrated plaque and the tumoro-ulcerative, are represented as well as various overlapping combinations. In addition, a small group of 51 patients with primary disease of lymph nodes accompanying or preceding the cutaneous involvement, so-called secondary lymphoma cutis, is included. The cutaneous manifestations of this group may mimic those of mycosis fungoides.

Mycosis fungoides presents a diagnostic problem in the early erythematous stage, even when generalized. Skin biopsy may show only a chonic dermatitis, and some of our patients with this tentative diagnosis have been treated for varying periods before histologic proof was forthcoming. Repeated and multiple biopsies, or typical response to electron therapy or progression to the next histologically definitive stage, may be necessary before the exact nature of the disease becomes evident.

Although the tumoro-ulcerative stage heralds approaching terminal disease, the erythematous (premycosis) and the infiltrated plaque stages may be commensurate

TABLE I

TREATMENT WITH LOW MEGAVOLT ELECTRONS (1951–1960)

		No. of Patients
Malignant Cutaneous Disease		400
Lymphoma cutis		271
Mycosis fungoides	220	
Lymphosarcoma	27	
Hodgkin's disease	13	
Lymphoma, not specified	5	
Reticulum cell sarcoma	3	
Leukemia	3	
Primary skin carcinoma		47
Basal cell	28	
Epidermoid	15	
Bowen's disease	4	
Skin metastases of breast carcinoma		28
Kaposi's disease		25
Miscellaneous		29
(Metastatic carcinoma from parotid, gingiva, tongue; melanosarcoma; hidradenocarcinoma; choriocarcinoma; Ewing's tumor; meningioma)		
Benign Cutaneous Disease		122
Generalized dermatoses		43
(Atopic, exfoliative, neurodermatitis; seborrheic psoriasis)		
Keloids		52
Miscellaneous		27
(Fox-Fordyce disease, furunculosis, granulomas, histiocytosis X, Hansen's disease, neurofibroma, neurogenic anhidrosis, papilloma, pemphigus, poikiloderma, undiagnosed, verruca vulgaris)		
Total		522

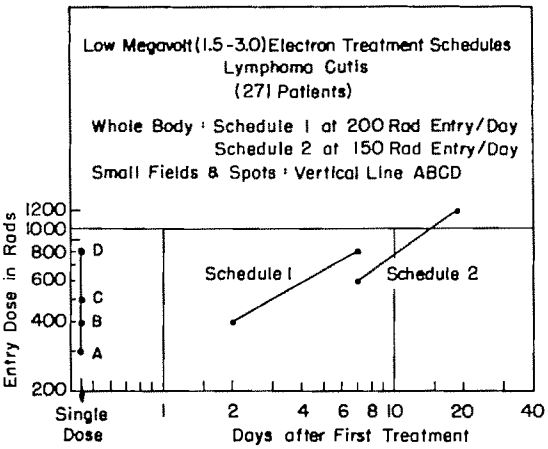


FIG. 3. Log-log plots of 2 empirically derived treatment schedules as guides in treatment of lymphoma cutis (mycosis fungoides). Schedule 1, at a daily entry dose of 200 rad to 2 opposing surfaces (of a total of 4) of whole body, was most commonly used and always taken to 600 or 800 rad. Schedule 2 represents daily entry dose of 150 rad, same surfaces, carried to minimum of 600 rad but not infrequently to 750 or 900 rad. Vertical line ABCD represents single dose treatments; point B (400 rad entry) always tried initially and used most commonly.

with many years of life. This implies the need for careful management in the essentially palliative therapy of this disease. We consider every patient in the first 2 stages to have a potential survival of years, and feel that conservative radiation therapy consistent with attainment of relief of the distressing cutaneous symptoms and appearance should be the rule. Complete evaluation of the patient with particular regard to the blood and marrow status is made. This implies close cooperation with other medical specialties, particularly the dermatologic and hematologic services. Since the majority of our patients are referred from widespread geographic areas, coordination is necessary with the referring physician who will carry the burden during the intervals between electron therapy.

A standard 4-field treatment of the entire body surface, with small field supplement (Fig. 3, schedule 1, B), is administered to the patient who has generalized cutaneous involvement in the erythematous, the infiltrated plaque and even the small nodular stages, with or without ulceration. A total dose of 600 or 800 rad, at 2.5 mev. is delivered in 8 to 10 days, at daily increments of 200 rad to 2 (of a total of 4) opposing body surfaces (Fig. 4). The anterior and posterior surfaces are treated alternately with the lateral surfaces on succeeding days. The face, eyelids, axillary, perineal and perianal regions are shielded during the transit of the patient beneath the cone when large surface treatments are given, but receive separate single doses of 400 rad once during the entire schedule. The eyes are always shielded. If eyelids are involved, internal eyecups are inserted after the conjunctivae are anesthetized, and a single dose of 400 rad is administered. A single dose of 400 r with 2 mev. roentgen radiation is administered to involved interdigital surfaces of toes and external auditory canals.

An alternative schedule of 4-field radiation (Fig. 3, schedule 2) at a daily rate of 150 rad may be used on occasions. This may be carried to final doses of 600, 750, 900 and rarely as high as 1,200 rad, depending on special circumstances, such as severity and extent of ulcerations, number and thickness of nodules and degree of regression during the treatment period. This schedule has been used in: (1) debilitated patients, (2) patients with demonstrated sensitivity to sunlight and ionizing radiations, (3) patients with involvement in areas of radiation dermatitis, (4) patients with widespread ulcerations, (5) some patients with poikilo-

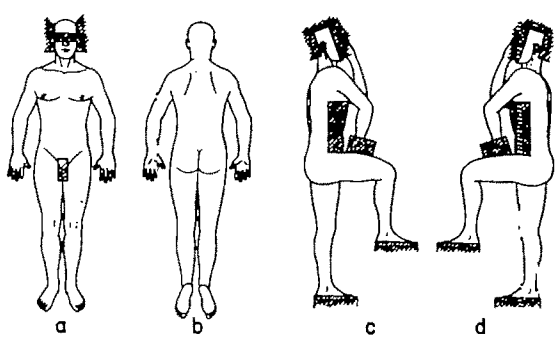


FIG. 4. Position and usual shielding of standard 4-field whole body surface electron treatment in lymphoma cutis. Surfaces *a* and *b* are alternated with *c* and *d* on successive days.

derma, (6) patients with large scarred areas with involvement, or (7) patients with unusually severe skin discomfort. In patients showing nodular tumors as thick as 2 to 3 cm., either schedule has on occasion been carried to final doses of 1,200 rad, at 2.5 to 4.0 mev. with complete regression. With either schedule, final dose will depend not only on the known or expected response for the lesion type, but also on geographic factors. To minimize the likelihood that the patient may have to return early from a long distance for "touch up" spot treatment of a few incompletely regressed lesions, we may strive for the 800 rad dose in schedule 1 or the 900 rad dose in schedule 2. The average patient in the first 2 stages will show healing about 2 weeks after completion of electron therapy. Nodular and tumoro-ulcerative disease may require up to 6 weeks for healing when dose and voltage have been adequate.

Patients were usually referred to us because of generalized skin involvement and after various conventional methods of therapy (superficial roentgen rays, steroids and chemotherapy) had failed to produce amelioration of appearance and symptoms. Only 10 patients (4 per cent) never required electron radiation of the entire body surface. This contrasts with 85 patients (38 per cent) who have received treatments only to the whole body surface. This latter group consists of patients who returned only when there was generalized or at least 50 per cent surface recurrence. The average number of treatment courses per year has been 2.4; some patients have required as few as 1 in 18 months and a few have had up to 7 in one year.

Small field and spot treatments with 400 rad single dose nearly always resulted in complete regression of lesions unless the tumors were unusually thick. In this situation we would repeat the spot dose and use higher voltage. Nodules 3 cm. thick have healed when 3.5 mev. was used although penetration of maximum dose would appear to be insufficient. Successively higher doses (Fig. 3, C, D) to small fields or spots usually no larger than 3 cm. may be needed in a few patients. This requirement for higher dose has heralded transition from mycosis fungoides to reticulum cell sarcoma. Since over 75 per cent of the patients entering the final phase of this disease do not return to us terminally, our data on this point are incomplete. Transition to reticulum cell sarcoma alone or mixed with lymphosarcoma occurred in 9 patients. The commonest transition terminally appeared to be to lymphosarcoma (24 patients); other transitions have been to Hodgkin's disease, lymphocytoma, lymphatic and monocytic leukemia and various combinations of these.

Alleviation of the distressing pruritus accompanying this disease has been possible in 98 per cent of patients treated. Unequivocally poor control of pruritus and lesions occurred in 3 patients with mycosis fungoides and in 2 who had leukemia as the primary or associated disease.

Eighty-nine patients (40 per cent) who had generalized cutaneous mycosis fungoides have survived from 2 to 7 years after electron therapy was begun. Twenty-three of these have had only 1 or 2 courses of electron therapy, and 4 have had from 4 to 11 courses, followed by no further recurrence for more than 2 years. Evaluation of the exact role of electron therapy in survival time is difficult and hazardous in this disease. This is because of the vagaries of the premycosis stage, the marked variation in survival of patients in the same age group and in apparently similar stages of disease, the difficulty in dating the onset of true histologic malignant lesions and the intrusion of other methods of treatment during intervals between electron therapy. Males predominate by a ratio of 5 to 2, although the disease pattern appears similar in both sexes. Analysis of our data suggests broadly that survival can be significantly extended, with comfort, by judicious use of electrons.

After the patient has entered the tumoro-ulcerative stage, there is advantage in combining electron therapy with steroids or chlorambucil, or both. Short courses of chlorambucil have been given to patients

who require frequent electron radiation (every 2 to 3 months) for whole body surface recurrence. It is our impression that this may actually defer recurrence by several weeks, or modify it sufficiently so that the number of treatments required per year is decreased. Electron therapy, however, remains the bedrock of any treatment program.

The largest number of treatment series given any patient has been 38, of which 5 were whole body surface and 33 spot and segmental treatments (from one-tenth to one-half the total body surface). After 6 years of electron therapy a patient showed generalized telangiectasia, scarring in previously ulcerated areas and areas of increased and decreased pigmentation. He finally succumbed to coronary thrombosis. Some patients have received more than 20 treatment courses. Two patients, still surviving, have had a total of 16 (13 whole body surface) and 17 (7 whole body surface) treatments, respectively. They show to a striking degree the problem of overlap presented by the technique described for whole body surface radiation using 4 fields. Both show an accentuated zone of telangiectasia along the anterolateral and posterolateral aspects of the trunk and thighs, where shielding of the junctions of the fields has been difficult. The overlap dose has been estimated to be between 6,900 and 7,600 rad when telangiectasia first became pronounced. Another patient with frequent isolated recurrences in identical or closely situated sites on the genitalia and perineum showed mild telangiectasia after an accumulated dose of 6,300 rad. We are now considering the use of a 6-field technique embodying a shifting overlap zone. This will not, however, solve the problem of numerous treatments that might be required for similar or nearby sites.

Depression of hematopoiesis occurred in 10 of the first 40 patients treated.[2] Some of these patients received much larger doses, up to 2,000 rad per treatment course, than subsequent patients treated by the schedules herein described. In addition, the accompanying roentgen-ray component was fourfold greater than it has been in the last 181 patients. In the latter group, the only hematologic abnormality that may be construed to be a result of electron therapy has been an occasional transient lymphopenia. Various degrees of marrow depression have been seen, but these have been the result of previous or interval chemotherapy and superficial roentgen ray therapy, or have occurred in patients with lymphomatous involvement of the marrow.

The rampantly fatal potentiality of this disease is exemplified by 43 patients who died within 1 year after 1 or 2 courses of electron therapy. Such a fulminant course usually accompanies transition of mycosis fungoides to one of the generalized lymphomas, with lymphatic, visceral and marrow involvement.

Electron therapy in the low megavolt range appears to be the ideal method of radiation for the skin manifestations of lymphoma cutis, particularly the mycosis fungoides variant. Barring those patients in whom the disease is fulminant, electron therapy in the majority of patients treated has successfully mitigated the often appalling and always distressing symptoms and appearances of mycosis fungoides for longer periods and more completely than is possible with any other single method presently available. Coupled with judicious employment of steroids and chemotherapy, the frequency of use of electrons may be decreased, thereby delaying the onset of radiation dermatitis which must inevitably ensue if the patient lives long enough and continues to have recurrences necessitating electron therapy. Unlike superficial roentgen therapy, repeated use of low megavolt electrons, as described, has not depressed the bone marrow. Radiation sickness does not occur irrespective of sites or size of surface area treated.

Skin Metastases of Breast Carcinoma

When breast carcinoma has metastasized, the skin manifestations may occasionally be the dominant aspect of the disease. Despite the application of hormonal

and chemotherapy, radiation may be a necessary adjunct treatment. Solitary nodules and small fields of skin recurrence are adequately handled with conventional and superficial radiation therapy. Our interest in applying electrons in this condition is based on the same factors that apply in the treatment of lymphoma cutis. The limited penetrability of electrons, which would protect the nearby underlying ribs, and the ease of covering large fields of involvement were persuasive.

The extent of cutaneous disease in 28 female patients varied from a small cluster of nodules up to 75 per cent of the thoracic circumference. In the majority (16 patients), either the right or left anterior half of the thorax was involved, with varying degrees of lateral extension. Radiation was delivered in the same manner as for lymphoma cutis. The uninvolved skin surface was shielded with masonite and the patient was passed beneath the treatment cone on the motorized couch, receiving electron radiation to the entire presenting involved surface. This implied that, because of the curvature of the thorax, 2 and sometimes 3 separate transits in 1 treatment might be required for complete coverage. By close shielding and changing the line of adjacent fields, overlap was minimized and never became a problem. Because of the severity and extent of disease elsewhere, we could not complete our empirical schedule (Fig. 5) by a rather wide margin in 12 patients. Nevertheless, 5 of these 12 patients were successfully treated, with complete regression of metastatic implants for a minimum of 6 months. Two of these 5 later showed new implants within the treated field and 1 at the very margins. It is difficult to evaluate the part that reseeding from foci outside the treated field plays in these cases.

Our empirical schedule (Fig. 5, ABC) shows 2 essentially different rates of treatment. Segment AB is for daily doses of 300 to 500 rad and is used for small cluster fields up to areas of approximately 20 by 20 cm. Only 3 cases are in this category. It is apparent that for small fields the treatment

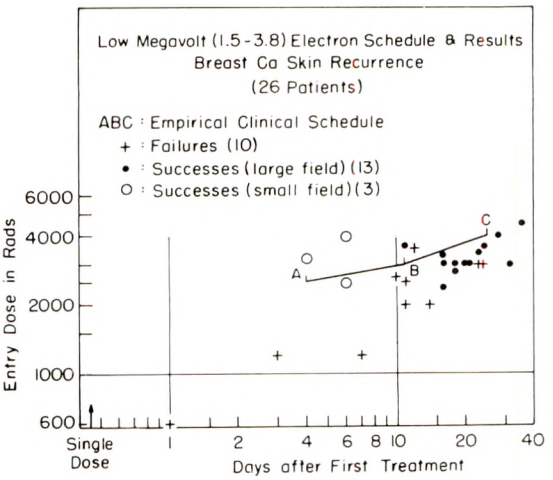


FIG. 5. Curve ABC represents our empirically derived clinical guide in the treatment of extensive skin surface fields involved by metastatic breast carcinoma. Daily entry dosage of 300 to 500 rad for small to moderate fields (up to 20 by 20 cm.) is represented by segment AB. Larger fields, up to three-fourths of chest circumference, are represented by segment BC where daily dose is 200 to 250 rad. Symbols show actual dose attained.

schedule is easily attained; also, the use of small fields usually implies less vigorously disseminated disease. These 3 patients received daily doses of 500 and 800 rad, and the largest field was 6 cm.^{2}

Line BC represents protraction at a slower rate because of large fields (over 20 cm.^{2}) and the daily doses here were 200 and 250 rad. The disease was much more active in these patients and the majority required from 14 to 35 days to complete a course. One patient who received 2,400 rad in 17 total elapsed days showed excellent response with complete healing; this patient had obtained satisfactory, though incomplete, regression of skin implants following administration of stilbestrol and this medication was continued during the electron treatment. The single failure above line ABC was a patient who had 2 courses of conventional roentgen ray therapy, totaling 4,800 r over a 5 month period, to a 10 by 10 cm. field which contained 2 ulcerations, 3 cm. and 2.5 cm. in diameter. The ulcerations had not healed despite this and adjunct hormonal therapy. An electron field

measuring 6 by 10 cm. was protracted through 13 treatments to 3,500 rad, with shrinkage, but the ulcers did not heal. Three and one-half months later, the ulcers coalesced and necrosis resulted. The patient died shortly afterward from disseminated disease.

The results of electron therapy were considered successful if complete regression took place and if the lesions did not recur in the treated field for a minimum of 6 months. This occurred in 15 patients, 1 of whom showed recurrence in the field at 7½ months. The other patients did not have skin recurrences but in many of them disseminated disease developed.

With daily increments of 300 rad to fields measuring up to approximately 20 by 20 cm., the skin reactions vary greatly. A few patients did not have significant skin reactions even at 3,000 rad. Others showed a brisk reaction within 10 days after a total dose ranging between 3,000 and 4,000 rad. When large nodules were present, some denudation occurred at the 4,000 rad level (Table II).

A range of 1.5 to 3.8 mev. was used in this series; 15 patients were treated with 2.5

TABLE II

SKIN REACTIONS WITH LOW MEGAVOLT ELECTRONS

I. *Normal Skin*
- A. Small treatment areas (keloid scars 1.5 by 10 cm.)
  1. No reaction from single dose of 1,000 to 1,500 rad at 2.5 mev.
  2. Transient blush (24 hours) in 1 week after single dose of 2,000 rad at 2.5 mev.
  3. Spotty desquamation and pigmentation or alternately no reaction at all, 4 weeks after single dose of 2,000 rad at 2.5 mev.
- B. 2 to 3 cm. areas
  Faint erythema 3 weeks after single dose of 1,800 rad at 2.5 mev.

II. *Hair*
- A. Scalp may epilate after 450 rad given in 1 week
- B. Beard may epilate after 900 rad given in 1 week

III. *Diseased Skin*
- A. Mycosis fungoides
  (Whole body surface fractionated treatment)
  1. Inflammatory areas begin to fade after 300 rad
  2. Infiltrated plaques begin to fade at periphery after 300 to 400 rad
  3. Plaques may begin to desquamate after 300 to 400 rad
  4. Pink blush to normal skin surrounding lesions after 300 to 450 rad
  5. Maximum erythema within 1 week after completion of full body dose to 600 rad; a few patients show no reaction; erythema is extremely variable
  6. Superficial ulcerations begin to epithelize after 400 rad
  7. Pruritus may temporarily increase hours after first dose of 200 rad in small number of patients
  8. Erythematous stage of mycosis fungoides usually shows complete healing 2 to 3 weeks after course of 600 to 800 rad
  9. Tumoro-ulcerative stage requires 4 to 6 weeks for complete healing
- B. Generalized nonspecific dermatoses
  (Whole body surface fractionated treatment)
  1. Skin weeping diminishes 48 hours after first dose of 150 to 200 rad
  2. Desquamation ensues soon after diminished weeping in short treatment course
  3. Desquamation may begin after first treatment in predominantly dry dermatoses
  4. Pruritus may increase transiently after first 150 to 200 rad dose
  5. General cutaneous discomfort may increase after first 200 rad dose and not after 150 rad dose
  6. Pruritus nearly always ameliorated before skin clears
  7. Total skin improvement (if it occurs) may require 3 to 8 weeks
- C. Breast carcinoma skin metastases
  (Large field fractionated treatment)
  1. Mild erythema at 1,400 rad dose
  2. Brisk erythema at 3,000 rad dose
  3. Erythema subsiding 7–10 days after 3,000 rad
  4. Slight residual erythema or milk tanning and flaky desquamation 4 weeks after 3,000 rad
  5. Slight denudation of tumor nodules after 4,000 rad
  6. Variable responses depend on:
     a. Size and type of tumor implants
     b. Presence of ulceration
     c. Presence of inflammatory changes
     d. Vascularity of mastectomy scar

mev., 7 with 3.0 mev., 3 with 3.5 mev., 2 with 1.5 mev., and 1 with 3.8 mev. The choice of voltage was entirely dependent upon the clinical appraisal of the thickness of the lesions. Except in 2 cases, none of the failures could be clearly construed as dependent on insufficient voltage. Nevertheless, it is possible that reseeding within the treated field might have been circumvented in 3 others, if it is postulated that recurrence resulted from nonirradiated tumor cells in lymphatic vessels on the pleural side of the chest wall, in which case penetrability was inadequate.

Electron therapy has become our primary radiation method of treating recurrences of metastatic breast carcinoma in the skin, and is particularly suited for large fields. Some further improvement in results is possible by the use of even larger fields, extended well beyond the visible and palpable borders of implants. Possibly 3.0 and 3.8 mev., rather than lower voltages, should be used on the supposition that tumor cells in lymphatic vessels on the inner aspect of the chest wall play an important part in reseeding the treated sites. The clinical dosage schedules appear adequate.

PRIMARY SKIN CANCER

The better cosmetic result expected from megavolt electron therapy and the relative ease of application of radiation with our apparatus and technique were the primary inducements to attempt to treat this group of lesions. Small spot fields, usually between 2 and 4 cm. in diameter, are easily managed by simply shielding with aluminum or masonite absorbers, or both, of appropriate size and shape. Actual treatment is accomplished during transit of the segment of couch and patient with the lesion centered beneath the cone. A single transit requiring a few seconds was sufficient for doses up to 800 rad.

Of the 47 patients in this group, 28 had basal cell carcinoma, 15 epidermoid carcinoma and 4 Bowen's disease. Excluding those with Bowen's disease, the histologic and follow-up data are adequate for analyzing only 28 of these cases. The empirical treatment schedule (Fig. 6, AB) was our guide for the treatment of both basal cell and epidermoid carcinomas. This coincides with 1,800 rad in a single dose, 2,500 rad in 3 days, 3,200 rad in 6 days, and 3,600 rad in 10 days, reaching 4,500 rad in 30 days. This entry dose represents the minimum tumor dose. The selection of voltage from 1.5 to 3.8 mv. was based on clinical appraisal of the thickness of the lesion.

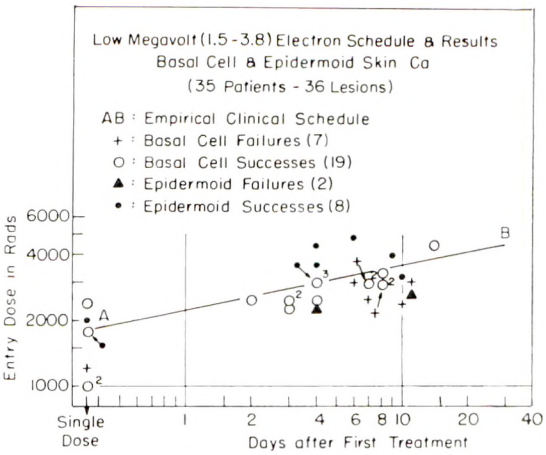


Fig. 6. Log-log plot of empirical treatment schedule (line AB). Symbols represent time-dose points actually obtained in each patient. Superscript numerals indicate number of patients at same point.

The plot of actual treatments on the log-log graph in Figure 6 presents some interesting factors. Despite our feeling that site, size and depth of infiltration are equally or more important than histologic differences in small skin cancers, we tended to use lower dosages in basal cell lesions. Exaggerated attention to cosmetic results caused us to shield too closely in our early cases. There were 9 failures; 2 of these patients had epidermoid and 7 basal cell cancers. In 7, the carcinomas were on the face; a review of these cases indicates that shielding was too close and may have had an important bearing on the failure in 4 patients. Underestimation of the depth of the lesion with insufficient penetration of dose was thought to play an important part in 4 other patients. In 2 patients treated at 1.5 mev., the

lesions remained healed for almost 1 year and then recurred, suggesting the possibility of regrowth from inadequately irradiated cells in the depth of the lesions.

One patient treated with 3.8 mev. electrons had a lumbar midline ulcerated mixed basal cell and epidermoid carcinoma which measured 3 cm. in diameter. It was thought worthwhile to attempt to obtain healing with electrons. Because of credence given to the "midline factor" with possible future difficulty with healing, a dose of 2,700 rad was given in 12 days. The ulcer appeared to heal satisfactorily, but $4\frac{1}{2}$ months later a recurrence, measuring 8 mm., was noted and excised. In another patient, who had a large, recurrent, basal-squamous cell carcinoma involving the entire graft site in the left temporoparieto-occipital region of the scalp and extending to the naso-orbital area, 3,600 rad was administered in 5 days, with complete healing.

Even with a wide scatter around the empirical dosage schedule (Fig. 6), successful results were obtained. In 1 patient who had 2 very superficial lesions, measuring 2.0 cm. and 3.0 cm. in diameter, on the abdominal wall, single doses of 1,000 rad at 1.5 mev. were given with complete healing. Histologic proof of malignancy was present in these 2 lesions.

The dosages given were readily tolerated at all protraction levels. When healing occurred, the cosmetic result was excellent whenever ulceration of the primary lesion was not extensive. After a single dose of 1,800 rad to a field of 2 or 3 cm., the height of reaction will be attained in about 2 weeks at which time the center of the lesion presents a thin eschar and is mildly pruritic. By the third week the reaction is reduced 50 per cent. At approximately 6 weeks the eschar will be gone, the center will show a tiny pit and the color of the skin will be almost normal. A 3 day protraction course leading to between 2,500 and 2,800 rad will show a similar sequence of events. With protraction to higher doses over 7 to 10 days, erythema will develop slightly later, crusting is likely to be less and healing occurs with minimal skin change.

Experience with 4 cases of Bowen's disease is insufficient for any definitive conclusions. Our ability to cover large surfaces homogeneously and expeditiously with electrons was tested in 1 patient who had widespread involvement of the entire trunk. Segmental field radiation was tried and he was given 1,650 rad over a period of 8 to 12 days; the entire involved surface was not treated daily. This dosage appeared to control the disease for 2 years. Subsequently, lesions developed at new sites as well as in areas treated previously. In another patient, 2 opposing lesions on the penile corona were given a total of 1,500 rad in 3 days. One site has remained healed, whereas the other required another 1,000 rad 3 months later.

Our limited experience with primary skin cancers suggests that 2 factors require special attention for successful results and at the same time optimal cosmetic results. Visual and palpable appraisal of margins and depth of the lesion may not be sufficiently accurate in some cases, resulting in undertreatment because of too great shielding or inadequate penetration. The choice of voltage implies that a greater burden is placed on clinical judgment concerning the thickness of the lesion, if optimal use is to be made of controllable penetration.

Kaposi's disease

The typical patient with this disease is a male showing violaceous nodules about 3 to 5 mm. in height, some singly, but more often coalescent over a wide area, on the lower extremities. The coalescent areas are likely to be distal to the knees, with scattered isolated nodules over the thighs.

A total of 25 patients, 23 males and 2 females, with this entity have been treated with electrons. We have found that the most satisfactory control of this disorder is obtained with a judicious combination of electron and 2 mev. roentgen ray therapy.

Isolated lesions have variable dose requirements; in general, however, a single dose of 800 to 1,000 rad is necessary to pro-

duce regression to a point that the lesions regress almost completely. Occasionally, this will be accomplished with a single dose of 500 rad but more often, the lesion will merely flatten, change to a brown color and remain in this inactive state. In more than half of our cases, the major surface of one or both legs from ankle to knee was affected, becoming coalescent in some areas. These were treated with daily doses of 200 rad, attempting to cover the entire circumference of the leg every 2 days, much as in lymphoma cutis. The dose was usually carried to 1,200 rad, although 800 rad was sufficient in several cases. Because of interdigital lesions and extremely thick lesions, the feet were usually treated to a similar dose with 2 mev. roentgen rays. We do not believe that this disease can be cured and thus prefer dosages which cause regression and relief of symptoms. Two mev. roentgen radiation is used to administer single or protracted doses to small lesions of the oral mucous membrane; 1,500 to 2,000 r has been administered to lesions in the lungs and gastrointestinal tract (stomach), with amelioration of pulmonary symptoms and cessation of bleeding.

In many patients the disease has been controlled for several years with this treatment scheme. Repeat treatments to extremities are necessary, although at highly variable intervals in many individuals. Recurrence does not appear as frequently as in lymphoma cutis. Isolated groups of active appearing violaceous nodules may show no activity for years after single doses of 500 or 1,000 rad. The disease advances with new outcroppings, however, and appears in other skin sites on arms, trunk and head.

In 4 patients so-called sarcomatous degeneration is known to have occurred. These lesions show decided resistance to the usual dose, as well as to much higher doses. They are often manifested visibly as exuberant, granulating lesions which may no longer show the purplish coloration. We have done less well here regardless of the dosage we have dared to give. Some partial regression occurs, but the limitations of radiation therapy are more obvious. One of these patients has been treated by isolation perfusion with chemotherapy, and inactivation and apparently complete healing occurred in some areas only to recur.

Occasionally, after a year or 2 of repeated treatments, the disease may enter a stage of inactivity and many lesions pass into a so-called fibrotic state. Some are in this state at the onset. This occurrence does not appear to be related to any unusually high treatment dose.

Varying degrees of edema of the lower extremities ensue eventually in all patients. Although this may be secondary to deep involvement, we find that the edema does not recede by radiation therapy alone, but does when the limb is elevated and elastic stockings are used. Radiation therapy is not indicated for the presence of edema alone.

In the over-all management of this disease, both electron and 2 mev. roentgen therapy have been necessary. Recurrence is unpredictable and may appear at sites immediately adjacent to areas treated with high doses. Our guiding principle is inactivation rather than any curative dose. Inactivating doses to small areas vary from 500 rad to 1,000 rad in single treatments. Eight patients have had follow-up studies for over 4 years; 4 of these have required 5 treatment visits in this period. Many isolated lesions have been treated with conventional therapy at home with satisfactory regression. These patients usually return for electron treatment when there are widely scattered lesions or large contiguous surface involvements. One male patient required 16 electron treatment series, chiefly to small fields, during a period of 5 years and now the disease is in a state of relative inactivity. Another patient, a woman, has had 20 treatment series in $2\frac{1}{2}$ years and the disease is in remission at present.

MISCELLANEOUS MALIGNANT TUMORS

Widespread skin involvement has been the criterion for using electrons in this varied group. Palliation is the primary aim in almost every patient. In 1 patient with

recurrent skin involvement from a parotid adenocarcinoma, 6,000 rad with 2.5 mev. electrons in conjunction with a similar tumor dose with 2 mev. roentgen rays was given to an abutting adjacent field on the neck and face, with healing and no recurrence for the past 6 years. Three patients with hidradenocarcinoma in fulminant widespread dissemination were given large field treatment dosages similar to those used for recurrences of breast carcinoma. Good initial response was obtained in 2 of these patients. Our limited experience with melanosarcoma does not justify any conclusions; this malignant tumor shows a highly variable response in different sites in the same patient. Dosage schedules of the order of 3,500 rad in 12 days with concomitant chemotherapy have been tried. However, even at these dosages, melanosarcomas continued to grow in some sites.

BENIGN DISEASE

GENERALIZED DERMATOSES

Patients with generalized superficial dermatoses were referred by the Department of Dermatology for whole body surface electron therapy. A total of 43 patients make up this group (Table 1), which includes atopic eczema, disseminated neurodermatitis, exfoliative dermatitis, psoriasis and a few less well classified erythrodermas overlapping these categories. Our basic reluctance to treat benign disease, in which response to radiation may be quixotic and ephemeral, was overcome by the disturbing clinical status of these patients. In the majority, the disease had become chronic, apparently progressive and in all instances disabling. Severe pruritus and cutaneous distress were the demanding symptoms. The skin was red, often edematous, excoriated, oozing and scaly. Some patients presented appearances similar to patients with mycosis fungoides.

The treatment schedules selected were entirely empirical and were altered according to clinical impression and hunches. The majority was treated by schedules similar to those used in lymphoma cutis. Some patients were given a dose of only 450 rad and some as high as 1,400 rad in 31 days. The voltage range was 1.5 to 2.5 mv. In 11 patients (25 per cent) the response was considered good to excellent, with amelioration of the severe pruritus and complete clearance of the eruption. Another 11 patients showed about 50 per cent improvement in the appearance of the skin and the pruritus was completely ameliorated. In a third group of 11 patients the response was poor, with minimal and variable degrees of improvement of the skin and the pruritus. In 2 of these the rash and itch worsened. In 10 patients follow-up data are inadequate for evaluation. Many of the patients showing response required continued close dermatologic care for maintenance of the improvement.

When no improvement was noted 1 week after administration of an initial dose of 400 rad, a longer protraction schedule was attempted. There is no evidence, however, that response ultimately was better with the higher total dosage and protraction. We now tend toward use of this longer schedule when the history suggests increased sensitivity to sunlight, and when the patient shows a particularly acute phase of eruption or any evidence of debilitation. Thus, an initial dose of 200 rad may be given to 2 opposing body surfaces on 2 successive days; if any evidence of exacerbation of eruption or cutaneous distress results, the dose is reduced to 150 or 100 rad and therapy given every 2 to 5 days. Another method is administration of 100 rad in 1 day over the entire body surface once a week up to 9 weeks.

A dramatic response in this group of generalized dermatoses occurred in 1 case following the use of a lymphoma cutis (schedule 1) treatment course; the eruption completely cleared and the pruritus disappeared in 2 weeks. In general, improvement tends to occur slowly, in from 3 weeks to 2 months. Whole body surface retreatments have been given to 7 patients (twice) on the basis of good response initially, followed by similar severe recurrence in 2 or 3

months. Although no evidence of marrow depression and no radiation sequelae have appeared, electron therapy seems to be of limited value for the benign generalized dermatoses.

KELOIDS

This benign condition, which is an atypical repair process of the skin, can at times tax the equanimity and acumen of the radiologist. In our experience there appear to be 2 main aspects to the keloid problem. One concerns the cosmetic aspect, and the second is concerned with the symptomatic aspects—pain, sensitivity, itch and tenderness. In about one-third of our patients, both cosmetic and symptomatic aspects were present (Table III). Keloids on the face, neck, sternal area and occasionally on the extremities presented primarily a cosmetic problem whether or not symptoms were associated. We believe these should be excised prior to irradiation if optimum results are to be obtained. For the symptomatic keloid, however, radiation is performed without excision.

Evaluation of the effectiveness of therapy, therefore, depends largely upon whether cosmetic or symptomatic factors prevail. Based on previous experience with superficial roentgen ray therapy, various dosage schemes were attempted with electrons (Table IV). The lowest effective dose that is unlikely to result in residual radiation skin change naturally is preferred for cosmetic reasons. This appears to be the 1,000 to 1,200 rad single treatment using closely shielded 2.5 mev. electrons. This is our policy of treatment for both previously excised and nonexcised keloids. For the latter, a higher dose may be more consistently effective; however, pigmentation may result. The large, nonexcised keloid may require some patience on the part of the patient and the therapist. Older keloids uniformly respond less readily than recent ones

TABLE III

LOW MEGAVOLT ELECTRON THERAPY IN KELOIDS
(54 Keloids in 52 Patients)

	Patients
The Problem	
Cosmetic: Commonly in females (face, neck, chest, extremities)	16
Symptomatic: Pruritic, tender, sensitive (almost any site)	20
Combined	16
Anatomic Sites	
Abdomen	14
Neck	12
Upper extremity	8
Chest	8
Lower extremity	6
Face	4

TABLE IV

LOW MEGAVOLT ELECTRON THERAPY
EXCISED KELOIDS

Effective Treatments					
One Dose		Two Dose*		Three Dose**	
Rad	Patients	Rad	Patients	Rad	Patients
800	1	1,000	1	2,400	1
1,000	2	2,000	1	2,800	1
1,200	2				
1,500	2				
2,000	2				

Noneffective Treatments			
One Dose		Two Dose*	
Rad	Patients	Rad	Patients
800	3	1,800	1
1,000	1		

Total: 18 Keloids, 17 Patients

NONEXCISED KELOIDS

Effective Treatments					
One Dose		Two Dose*		Three Dose**	
Rad	Patients	Rad	Patients	Rad	Patients
800	2	1,800	1	3,500	1
1,000	8	2,000	9	4,000	1
1,200	3	2,300	1	4,500	1
1,500	3	2,400	1		

Noneffective Treatments			
One Dose		Two Dose*	
Rad	Patients	Rad	Patients
450	1	2,000	1
500	2	2,500	1
800	2		

Total: 36 Keloids, 35 Patients

* Second dose delivered in 6 to 8 weeks.
** Three doses delivered over 3 to 5 months' time.

(3 months or less). Although there is no chance of reducing the size of an old keloid by radiation other than to flatten it, redness and symptoms may be improved to the satisfaction of the patient.

In 18 excised keloids, good cosmetic results were obtained in 13 patients with dosages of 1,000 to 2,000 rad. In 36 non-excised keloids, good symptomatic results were obtained in 26 patients using a similar dosage range. Five other patients responded well with doses in the 2,300 to 4,500 rad range, fractionated.

Our present plan for all patients with keloids is to administer a dose of 1,000 rad at 2.5 to 3.0 mev. and recheck their status in 6 to 8 weeks. At that time another similar dose may be administered if there is evidence of only partial regression of symptoms or thickness and redness of a non-excised keloid, or evidence of some keloidal growth in the excision scar. Treatment is administered with close shielding within 1 to 3 mm. of the keloidal or excised scar margins.

MISCELLANEOUS BENIGN LESIONS

A few isolated skin oddities, one each, were locally benefitted by electron therapy. Leishmania tropica, measuring 1.5 by 2.0 cm. was treated by 1,200 rad in 7 days; scattered patches of scaly eruption (considered to be an allergic phenomenon) secondary to Hansen's disease, on the chest, forearms, buttock and trunk responded to a total dose of 2,000 rad in 4 treatments. Three patients with granuloma annulare on the hands responded to a single dose of 800 rad as well as to 3 consecutive treatments totaling 1,200 rad.

CONCLUSIONS

Experience with low megavolt electron therapy over a 10 year period on 522 patients has been described. This new modality appears to be the ideal radiation procedure for the management of radiosensitive skin lesions involving large areas including up to the whole body surface. The following factors contribute to this performance: (a) the electron dose can be applied readily and quickly to large areas; (b) the penetration of the electrons can be adjusted to the thickness of the lesion with consequent sparing of underlying normal structures; and (c) the resultant low integral dose avoids adverse side effects.

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We wish to acknowledge the important radiation therapy contributions made during the first 2 years of this program by our former colleague in radiology, Dr. Hugh F. Hare, now at the Los Angeles Tumor Institute. We are indebted to many of our clinical colleagues, particularly Dr. Donat P. Cyr (hematology) and Dr. John L. Fromer (dermatology), for their contribution to the clinical care of the majority of these patients.

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10 MEV. BETATRON ELECTRON BEAM THERAPY ADAPTED TO A CASE OF MYCOSIS FUNGOIDES

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MYCOSIS fungoides is a variant of malignant lymphoma of the skin and may involve the entire body surface without giving rise to lymph node or visceral metastases. The lesion resembles a focus of lymphosarcoma in the dermis, then the epidermis becomes infiltrated and thickened. In the later stages tumors up to 1 cm. or more in thickness occur and secondary infection is common. As the usual clinical course is prolonged over many years, lesions in various stages of evolution generally coexist.

Mycosis fungoides is very radiosensitive. Individual lesions disappear and the appearance of the skin often returns to normal after radiation therapy with doses in the range from 200 r minimum to 800 r maximum,[2,3,5,13] but the tendency for the development of new lesions and for recurrence of lesions in irradiated areas makes a fifteen to twenty year follow-up mandatory before the radiocurability of mycosis fungoides can be evaluated. However, treatment of mycosis fungoides is not usually given with intent to cure, but for the relief of pruritus, disfigurement, and secondary infection.

The use of low-voltage roentgen irradiation is simple and customary for the treatment of individual lesions or small areas, but more consideration should now be given to the fact that this quality of radiation is unnecessarily penetrating for surface tumors or plaques, and that repeated treatments are likely to be required for many years.

Since various qualities of nonpenetrating radiation are readily available, *e.g.*, radioactive phosphorus, strontium or yttrium[7,8] and electron beams of various energies,[15] it would seem preferable to use them even for individual lesions.

When the entire body surface is involved, roentgen ray therapy is quite unsuitable, for the unnecessary irradiation of deeper tissues may cause lasting depression of the blood-forming tissues and of other radiosensitive organs.

The systemic use of radioactive isotopes is likewise limited; with radioactive phosphorus, for example, even though there is some selective uptake by the lesions, some damage to the bone marrow cannot be avoided. Thus for whole body surface irradiation, as well as for individual lesions, a beam of electrons of suitable energy is best.

Wright, Granke and Trump[13] devised an excellent method of irradiating the skin of the whole body with an electron beam from a Van de Graaff generator; the electrons emerged from a narrow slit over the width of body while the patient was moved from head to foot across the beam on a motor-driven table at such a rate that 200 r was given to the whole skin. In the course of a week's treatment, a lesion dose of 800 r was given with resultant complete disappearance of all the lesions.

The maximum penetration in tissue of 2 mev. electrons is 10 mm., and 80 per cent of the energy is absorbed in the first 5 mm. so that a 2 mev. generator is the best apparatus to use in the treatment of most cases of mycosis fungoides. However, electron beam generators operating at higher voltages, such as the betatron, can be used to give the same absorption characteristics by interposing a polystyrene absorber of suitable thickness between the source and the skin (Fig. 1). With our betatron unit operating at 10 mev. and with a 3.85 cm. thick polystyrene plate, the relative doses on the skin surface and at 3 mm., 5 mm., 10 mm. and 20 mm. are 100 per cent, 59

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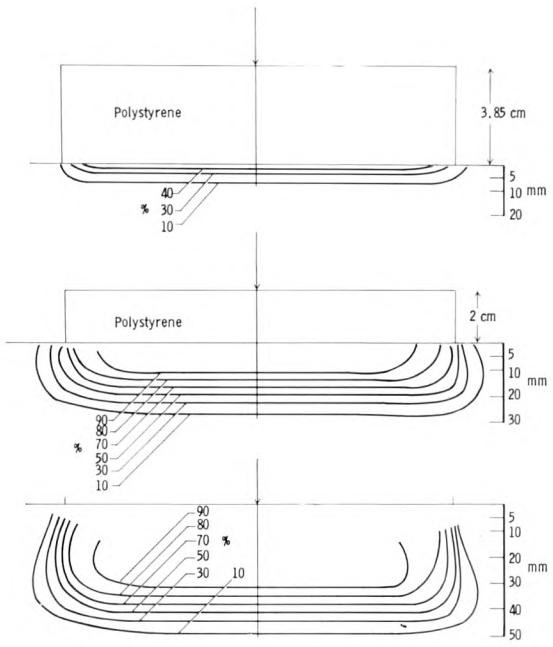


FIG. 1. Isodose curves for 10 mev. electron beam; field size 15×15 cm.

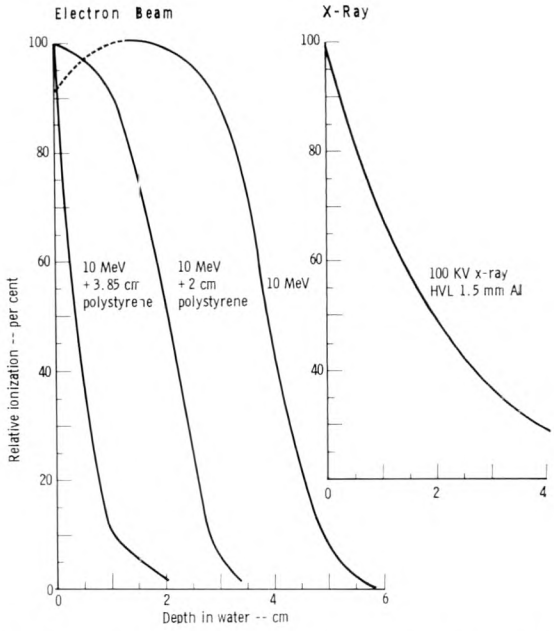


FIG. 2. Comparison of absorption in water of low voltage roentgen ray beam with electron beam.

per cent, 41 per cent, 11 per cent and 2 per cent respectively.

Depth dose curves of 100 kv. roentgen rays and of 10 mev. electron beams without an absorber, with a 2 cm. polystyrene plate and with a 3.85 cm. polystyrene plate are compared in Figure 2.

REPORT OF A CASE

The patient, a forty-six year old white male, was described in a previous publication.[9] He had a five year history of severe pruritic dermatitis, and had been a difficult diagnostic and therapeutic problem. Repeated skin biopsies had shown only nonspecific dermatitis until October 8, 1960 when a dense polymorphous mononuclear infiltrate was seen,

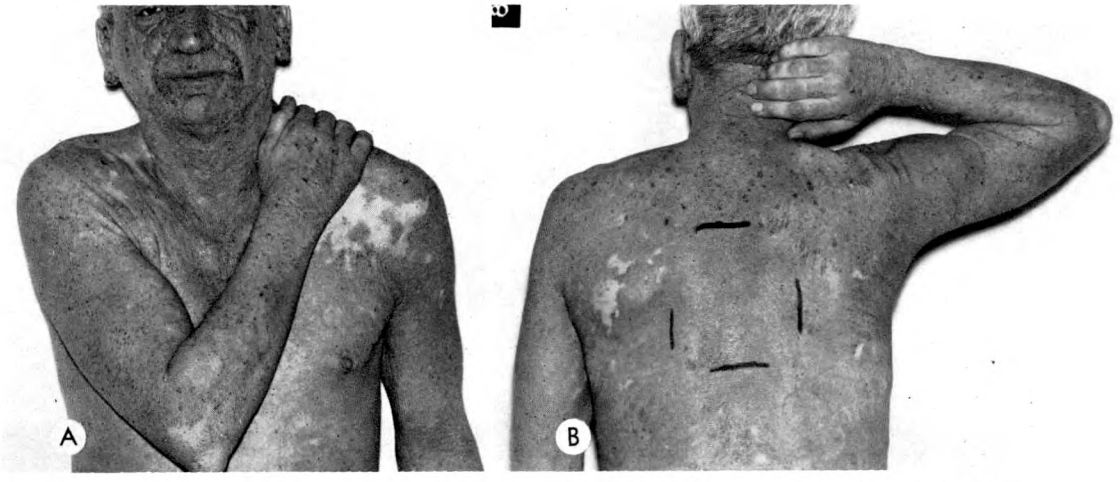


FIG. 3. (*A* and *B*) Appearance of patient at beginning of treatment on March 17, 1961.

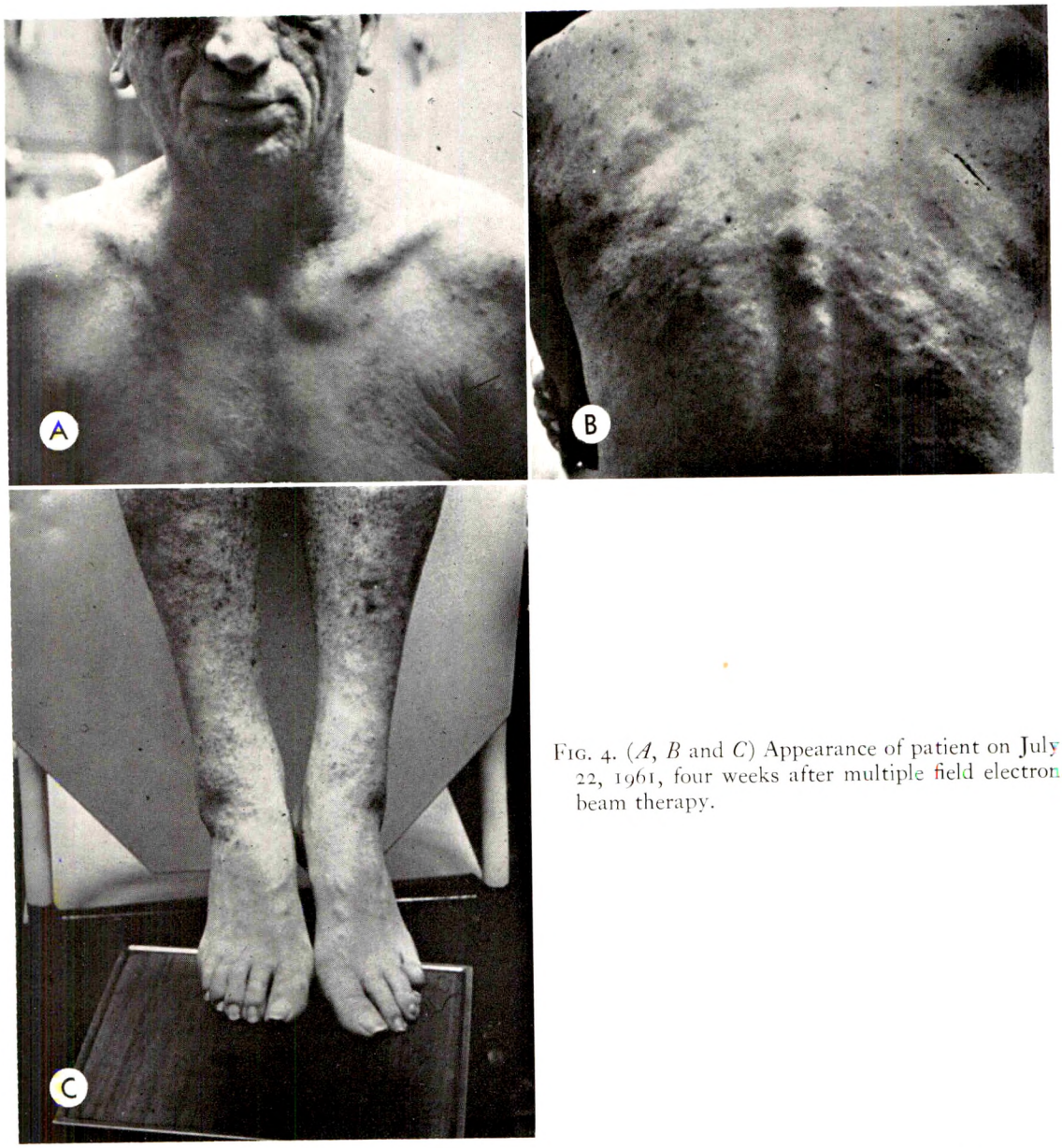


FIG. 4. (*A*, *B* and *C*) Appearance of patient on July 22, 1961, four weeks after multiple field electron beam therapy.

compatible with a diagnosis of mycosis fungoides. It had been found that 150 r of 90 kv. roentgen rays (0.9 mm. Al half value layer) had no effect on the lesions, but that 300 r was followed by complete resolution in two weeks. He was referred to Memorial Hospital for electron beam therapy as an out-patient.

Figure 3, *A* and *B* shows the patient at the beginning of treatment in March, 1961; Figure 4, *A*, *B* and *C* shows him on July 22, 1961, four weeks after completion of treatments. On his first visit, the two recently irradiated areas showed smooth skin (Fig. 3, *A* and *B*) with a slight yellowish tinge; otherwise the entire skin was covered with reddish lesions varying in thickness from 3 mm. to 5 mm., and with multiple excoriations on all accessible areas. The infiltration of the eyelids prevented closure and there was continuous tearing. A serous discharge from the ears was noted, but there was no overt infection or ulceration. The presence of palpable lymph nodes in the neck, axillae and groins was consistent with the generalized chronic dermatitis.

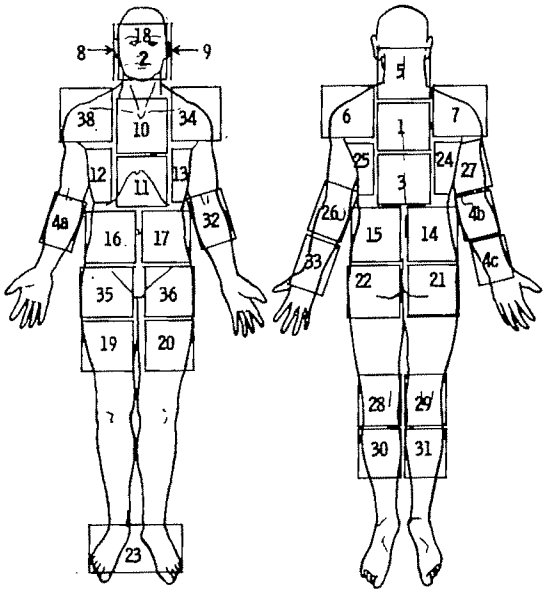


FIG. 5. Diagram of multiple fields used for electron beam therapy of generalized mycosis fungoides.

The rest of the physical examination was normal, as were the blood and bone marrow studies and the chest roentgenograms. In spite of the intolerable itching and loss of sleep, the patient's physical condition appeared good but his mental attitude was depressed and very negative.

Treatment. As we did not have facilities either for moving the patient under the betatron beam or for moving the beam across the patient, several 15×15 cm. areas were treated at each weekly visit until the entire skin surface had been covered (Fig. 5). When the eyes were in the field, the lens was protected by a polystyrene cylinder 6 cm. long and 1 cm. in diameter (Fig. 6).

The usual single dose given was 293 r at 3 mm. depth and more than half the areas so treated showed satisfactory regression of the lesions without recurrence during the subsequent weeks of observation when other areas were being treated. In five areas to which this dose was delivered and where there was no sign of regression one week later, a second dose of 293 r was given so that to these areas 586 r was given in eight days, but in none of them was complete regression of lesions obtained. During the patient's total period of attendance of fourteen weeks, second or third doses were delivered to other areas, but, as can be seen from the treatment summary in Table 1, there was no clear correlation between dose and response of the lesions or their subsequent recurrence.

In the later weeks, to two areas single doses of 500 r and to two areas single doses of 600 r were delivered. Within four weeks all four areas were clear of lesions. It seemed that lesions on exposed areas such as the face and hands responded less satisfactorily than lesions on covered areas such as the back and feet.

However, in spite of the many areas of clean skin where there had been none before treatment, the patient's complaint of intolerable itching was not noticeably alleviated. Treatment was discontinued before the optimum dose could be determined empirically, but our data suggest that a tissue dose of 500 r of 10 mev. electrons with a 3.85 cm. polystyrene absorber was equal to a tissue dose of 285 r of 90 kv. roentgen rays.

The patient is presently being treated elsewhere (Lahey Clinic, Boston), where he receives whole body electron beam therapy with a Van de Graaff generator at 2 mev. A comparison of the two modalities—multiple fields treated with single doses, and the whole body treated with fractionated doses—should be of interest in evaluating the best method.

All the above doses were calibrated in roentgens with a Victoreen ionization chamber.[6]

SUMMARY AND CONCLUSIONS

A case of mycosis fungoides of the whole body surface is reported. Electron beam

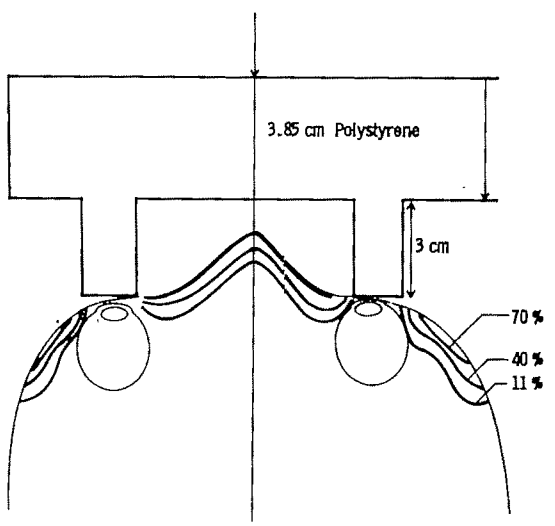


FIG. 6. Complete shielding of the lens of eye in electron beam therapy of face.

TABLE I

TREATMENT SUMMARY (Fig. 5)

Treatment	Dose (r) at 3 mm. Depth	Response	Part of Body
1	293 r, 2 wk. later 293 r and 2 wk. later 600 r	+	back
2	326 r, 4 wk. later 326 r and 8 wk. later 500 r	o	face
3	293 r in single exposure	+	back
4a	293 r in single exposure	+	arm
4b	293 r in single exposure	+	arm
4c	293 r in single exposure	+	arm
5	293 r, 1 wk. later 293 r	o	neck
6	293 r in single exposure	±	shoulder
7	293 r in single exposure	±	shoulder
8	326 r in single exposure	o	side of face
9	326 r in single exposure	o	side of face
10	293 r in single exposure	+	chest
11	293 r in single exposure	+	chest
12	293 r, 1 wk. later 293 r	±	chest
13	293 r, 1 wk. later 293 r	±	chest
14	293 r, 1 wk. later 293 r	±	back
15	293 r, 1 wk. later 293 r	±	back
16	293 r in single exposure	+	abdomen
17	293 r in single exposure	+	abdomen
18	326 r, 3 wk. later 326 r and 5 wk. later 500 r	o	forehead
19	293 r in single exposure	o	thigh
20	293 r in single exposure	o	thigh
21	293 r in single exposure	+	buttock
22	293 r in single exposure	+	buttock
23	293 r in single exposure	+	feet
24	293 r in single exposure	±	back
25	293 r in single exposure	±	back
26	293 r, 4 wk. later 500 r	±	arm
27	293 r, 4 wk. later 500 r	±	arm
28	293 r in single exposure	+	thigh
29	293 r in single exposure	+	thigh
30	293 r in single exposure	+	leg
31	293 r in single exposure	+	leg
32	293 r in single exposure	±	arm
33	293 r in single exposure	+	arm
34	293 r in single exposure	±	shoulder
35	500 r in single exposure	+	groin
36	500 r in single exposure	+	groin
37	500 r, 2 wk. later 600 r	+	hip
38	600 r in single exposure	+	shoulder
39	600 r in single exposure	+	hip

+ Complete regression.
± Incomplete regression.
o No regression.

therapy was given to 15×15 cm. areas until almost the whole body surface had been covered over a period of fourteen weeks, using a betatron unit operated at 10 mev. with a 3.85 cm. thick polystyrene absorber.

The depth dose distribution of this beam was 59 per cent of the surface dose at 3 mm. depth and 41 per cent at 5 mm. depth. Comparison of single doses of 293, 326, 500 and 600 r at 3 mm. depth on different areas suggests that a dose of about 450 r at 3 mm. depth was required for complete regression of lesions in this case, although a dose of 300 r at 3 mm. depth caused complete regression in more than half the areas so treated. Retreatment of an area given an inadequate dose one or more weeks later with the same dose also failed to effect a response. In retrospect it would probably have been better to have used only a 3 cm. thick polystyrene absorber with the 10 mev. electron beam to ensure an adequate dose at 5 mm. depth.

Treatment of the whole body surface through multiple 15×15 cm. areas is tedious and time consuming, and the method developed by Wright, Granke and Trump, in which the patient is moved across a slit-beam, seems preferable.

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PRODUCTION AND PROPERTIES OF HIGH ENERGY ELECTRONS FOR THERAPY*

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THE Memorial Sloan-Kettering betatron was adapted for external electrons in 1955 and treatment of patients commenced in June of the same year. This report describes apparatus developed for this use and the features of the dose distributions available with these high energy electrons.

In operation with electrons, the betatron has proved to be as reliable as with roentgen rays; there have been no maintenance problems and patient therapy and experimental irradiations have proceeded on a regular schedule with minimal interruption (2 days treatment time in 6 years). The machine is used mornings for patients, and afternoons for experimental work.

The energy of the electron beam is easily adjustable from 5 mev. to 24 mev. The ease with which the energy of the electron beam can be changed and the ability to adjust the direction and location of the betatron are valuable assets in its clinical application. The field sizes are determined by lucite and aluminum collimators which range in size from a 3 cm. diameter circle to a 15 cm.×15 cm. square, and include assorted rectangular fields ranging up to 24 cm.×15 cm. The dose distribution characteristics are distinctively different from those produced by roentgen ray beams. The planning of treatments also requires different solutions in obtaining a clinically acceptable dose distribution.

Experience in the development and clinical application of high energy electrons in the 2 mev. range,[14] 6 mev. range,[3] and up to 24 mev.[4,9,11] has been described in the literature. This report presents the experiments, equipment design and construction, and procedures carried out in the development and therapeutic application of a movable betatron as a source of high energy electrons. Details of the apparatus necessary for the extraction of the high energy electrons together with the control of their energy, field shape, intensity distribution, and monitoring are discussed. The measure of both exposure dose and absorbed dose is described. Extensive measurements of isodose contours in water for a variety of energies, field sizes, angles of incidence and wedges are presented. The combination of these distributions produced by assorted energies, field size, and frequency of use is also described.

BETATRON AND ELECTRON ACCELERATION

The Memorial Sloan-Kettering betatron is an Allis Chalmers model 24 RCM machine (Fig. 1). To adapt such a machine for electron beam therapy requires extraction of the beam[12,13,15] and the use of considerably different methods of collimation and beam compensation than are employed with roentgen rays.[10] Changes in the monitoring and electronic circuitry are also necessary. The solution of these technical problems described here results in a relatively simple and reliable electron therapy unit. Some of the features incorporated are: (1) a simple method of control of electron energy and insurance of a uniform intensity field at all energies and field sizes; (2) a constant patient skin distance and a set of interchangeable cones with known isodose curves; and (3) a light localizer which projects a crosshair shadow at the center of the field on the patient.

A cross section of the betatron when

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Presented at the Fifty-seventh Annual Meeting of the American Roentgen Ray Society, September 27, 1956, and brought up to date in December, 1961.
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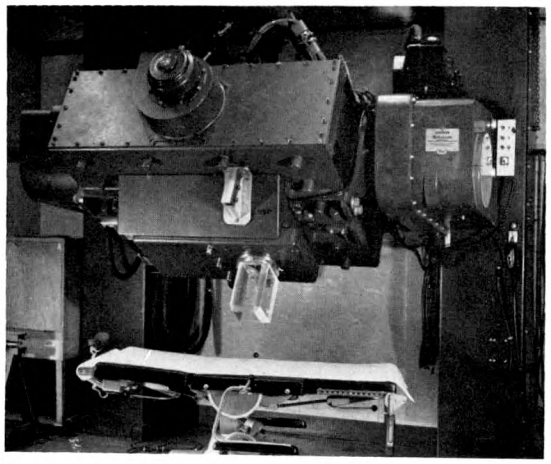


FIG. 1. Betatron as developed for electron beam therapy in treatment position.

used for electrons is shown in Figure 2. This betatron delivers a beam variable from 5 mev. to 24 mev. in 180 pulses per second. The electrons are first accelerated from a filament in the injector assembly byapplication of a 30 to 60 kv. potential negative pulse. They are accelerated in a circular orbit by the changing magnetic flux within their orbit. Their mean orbit radius is fixed at a distance where the magnetic flux density is half the average flux density over the mean orbit area.[5,6,7] This is adjusted for maximum output by shimming of the gaps in the magnetic circuit. After injection the electrons oscillate about the mean or equilibrium orbit. To minimize the quantity of electrons lost from their orbits by interaction with the injector and "peeler" (magnetic shunt), the individual orbits are caused to contract toward the equilibrium orbit at injection time through the application of a current pulse in a special coil on the pole faces.[1] For roentgen ray donuts the output is considerably improved by contraction and with electron beam donuts little output is obtained without it. This is due to the disturbance in the magnetic field

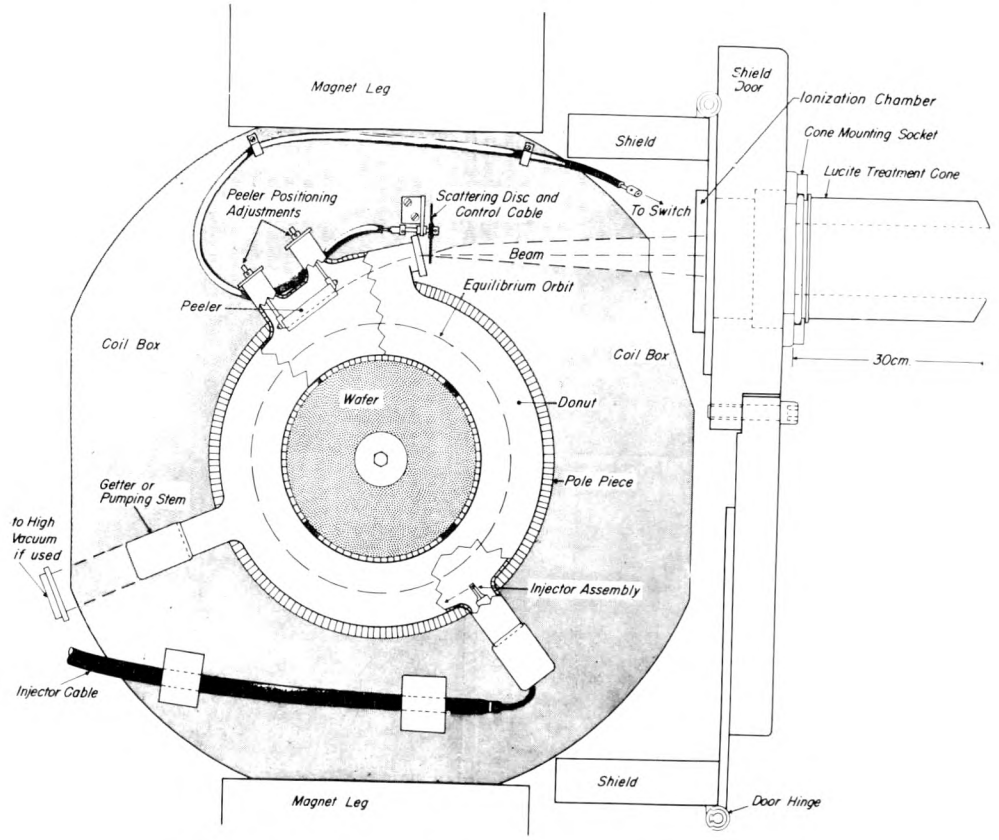


FIG. 2. Cross section of betatron in plane of orbit, top view.

caused by introduction of the magnetic shunt (Fig. 2) which is used to extract the electrons.

EXTRACTION OF ELECTRONS

As the electrons are accelerated in their orbits, the radius of the equilibrium orbit may be suddenly increased by application of a current pulse through a coil mounted on the poles. The electrons spiral out and pass through the field free region inside the magnetic shunt or "peeler" and escape tangentially from the equilibrium orbit. The time at which the expansion occurs may be anytime during the acceleration cycle. In this betatron, the electrons are expanded at the time of maximum energy and the electron beam energy is adjusted by controlling the maximum current applied to the betatron magnet. On the basis of experience with the University of Illinois Medical betatron which used fixed magnet amplitude and variable expansion timing for precise energy control, it was deemed desirable to modify the commercial contractor circuit so that the contraction pulse current was negligible as soon as the electrons approached energies of a few mev. This modified circuit is shown in Figure 3. Reduction of the capacitor size makes the contraction timing quite critical. The original circuit kept the beam somewhat contracted until maximum energy was reached, and interfered with expansion sufficiently to change beam direction with energy and limit extraction of the beam. This problem does not occur with maximum energy expansion.

The magnetic shunt consists of a stack of laminations of high saturation density magnetic alloy, usually Permendur. A slot runs through it lengthwise and provides a field free region in which the electrons are shielded from the guiding magnetic field. It is desirable that the shunt be positioned to ±.01 inches for maximum output. The following procedure was found helpful in the initial adjustment of the shunt:

(1) Provided that the magnet shimming is approximately correct so that an electron beam can be accelerated within the confines of the donut, it should be possible to observe some external beam if the shunt is not located at such a small radius that it destroys the orbit, or at such a large radius that the electrons fail to reach the shunt or strike the injector instead. It is not essential that the shunt be exactly tangential or in the orbit plane when adjusting to establish the existence of the electron beam. When first looking for a beam, an ionization survey meter viewed remotely is used—not a Geiger Müller counter since it will count only 180 pulses per second.

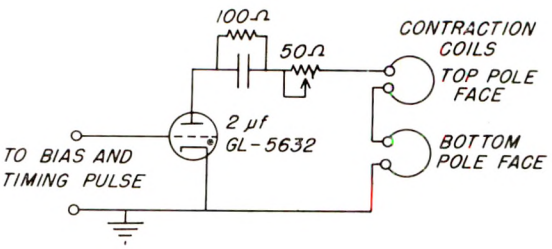


Fig. 3. Modified contractor circuit.

(2) Trial and error adjustment of the shunt to maximize the output will, with the following exception, lead to a proper planar and tangential alignment. When the shunt is too high or low and is at too great a radius, a maximum in output may sometimes be obtained due to scattering from the upper or lower lip of the peeler. A film (dental size is convenient) exposed to the beam at the donut window shows a very diffuse uncollimated beam in this case.

(3) Proper magnet shimming is mandatory for maximum output.

(4) Shunt adjustment should be done with the magnet energy rather high, say 20 mev., since adjustment is less critical at lower energies.

Due to the fringing magnetic field, the electron beam spirals slightly after leaving the shunt. This results in the electron beam being displaced about 10 cm. with respect to the roentgen ray beam position. The same opening in the shielding door was used in this case but the door was moved 4 inches (Fig. 4). With the dual roentgen ray

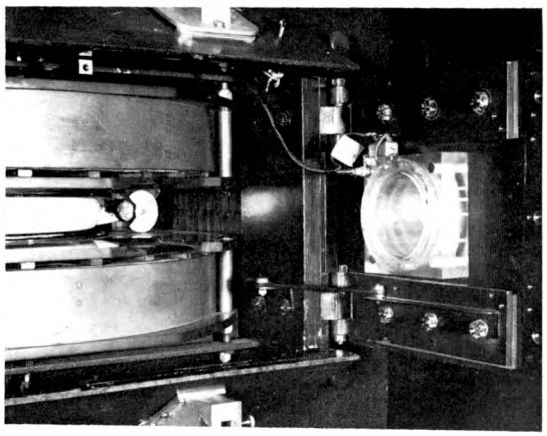


FIG. 4. Front of betatron with shield door open showing left to right: donut, scattering wheel, and monitoring ionization chamber which covers the hole in the door.

and electron beam donut, one beam emerges from each side of the donut through its own hole in each shield door.

FIELD COMPENSATION

The divergence of the electron beam after passing through the shunt is insufficient to provide uniform intensity over the area of the collimators at the treatment distance of 90 cm. from the center line of the betatron. A feasible means of spreading out the beam is to pass it through a thin foil scatterer. The loss of energy of the beam in the scatterer should be proportional to the electron density (electrons/cm.^{3}) of the foil; and the angle of deflection of electrons scattered by the foil should be proportional to the square of the atomic number. Thus, maximum scattering with minimum absorption is accomplished with high atomic number foils. This also maximizes the amount of bremsstrahlung produced in the scatterer, but since the total energy lost from the beam in the scatterer is quite small, the roentgen ray contamination of the electron beam is not of clinical significance; measurement shows this to be about 1 per cent.

The scatterers are mounted on a 4-step circular wedge with the following in each quadrant: (1) open; (2) 0.022 inch Al; (3) 0.022 inch Al+0.055 inch Pb; (4) 0.022 inch Al+0.010 inch Pb. These were designed for approximately 22 mev., 15 mev., 5 mev., and experimental use, respectively. In practice, the output of the betatron is so high in the 15 mev. region that scatterer No. 3 is often used in place of scatterer No. 2. This has little effect on the isodose contours since there is adequate scattering in either case.

A flexible cable is connected to the shaft of a 4 position rotary switch mounted on the front of the betatron. The switch index also positions the scattering disk while the switch contacts operate remote indicator lights on the control panel. This assembly is shown in Figure 4.

MONITORING

Monitoring is accomplished through a thin wall transmission ionization chamber (Fig. 4 and 5). Three sheets of 0.001 inch dural foil are mounted tightly stretched in a lucite frame which spaces them 2 mm. apart. The collection voltage is applied to the outer plates while the central collection electrode is connected to the amplifier. Two outer 0.005 inch mylar sheets protect the plates from mechanical damage. A dose rate of 1 rad per second at the cone produces a monitoring current of 10^{-8} amperes.

An integrating amplifier was developed to have high accuracy and wide range (Fig. 6). Essentially, the ionization current is chopped by a 60 cps chopper and the resultant signal amplified by a Brown high impedance amplifier. The output drives a 2 phase Diehl servo motor connected to a tachometer generator and to a Veeder-Root counter. The output of the tachometer generator is connected to a multiple scale voltmeter (rate meter) and through a 100 megohm feedback resistor to the input. Voltages of the order of 5 volts at motor speeds of 1,000 rpm are usual. The counter tabulates the dose. With this type of circuit, care must be taken that the motor is not driven close to synchronous speed as it will overload and fail to register the dosage given. This occurs at dose rates above 300 rads per minute for this design, which is triple the maximum rate used in treatment.

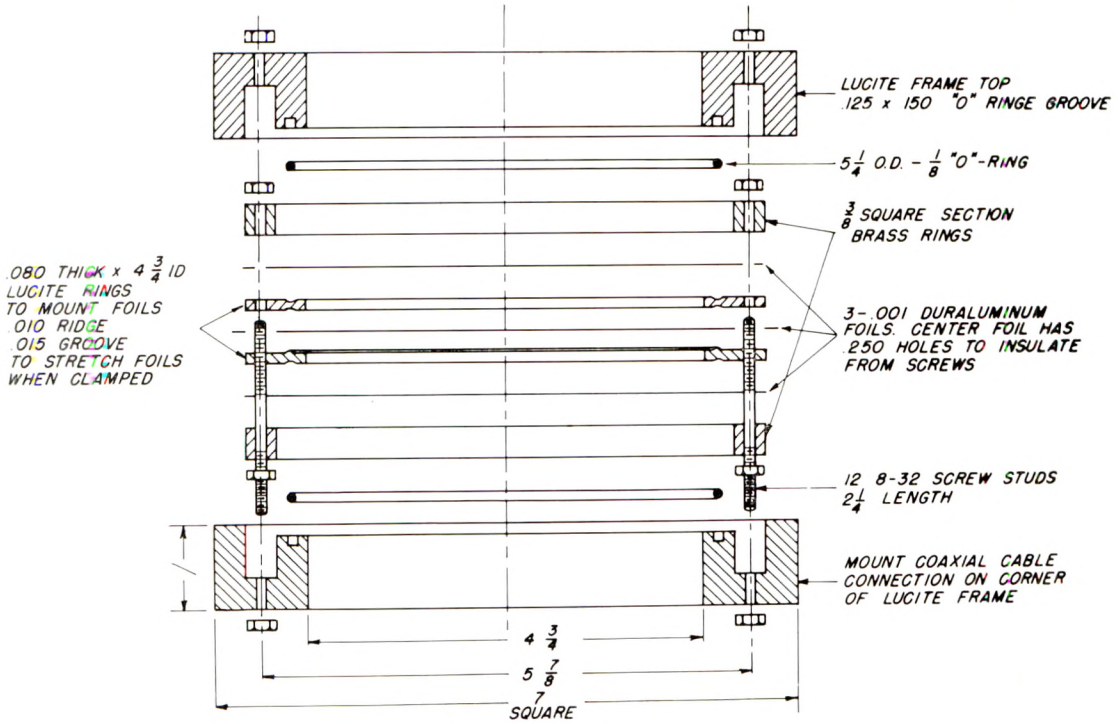


FIG. 5. Exploded view of the monitoring ionization chamber.

COLLIMATION OF THE BEAM

The collimators are made either of lucite with walls a minimum of $1\frac{1}{4}$ inches thick, or of aluminum with walls $\frac{3}{8}$ inch thick. Combination cones of both lucite and aluminum are also used. Aluminum collimators produce a slightly better depth dose distribution but this effect is not sufficiently pronounced to be important for clinical application (Fig. 7). These collimators carry a pair of crosshairs (black nylon suture or fishline) whose shadow is projected onto the patient by the localizer light. The optimum position is about 2 inches from the end of the end of the collimators where it cannot touch protruding tissue. The ring shaped socket for holding the collimators on the betatron allows 360° rotation as well

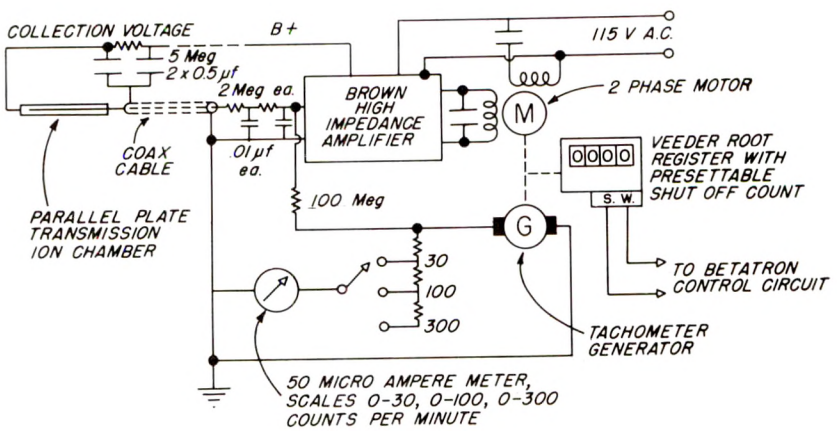


FIG. 6. Schematic diagram of the integrator circuit.

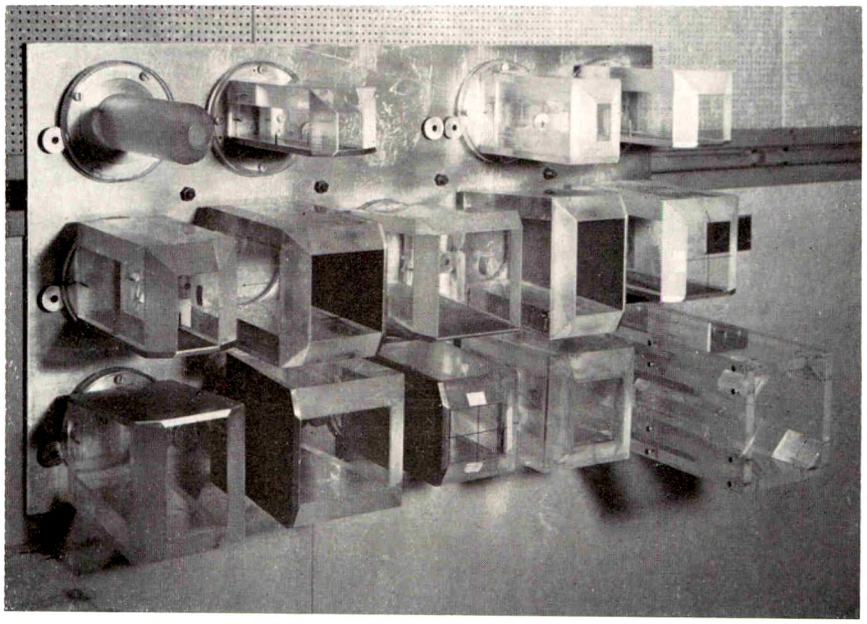


FIG. 7. Some of the collimators on a storage rack.

as rapid interchange and is equipped with a protractor (Fig. 8). This ring and the mounting rings on the collimator bases are satin-chrome plated brass.

LIGHT LOCALIZER

Figure 8 shows the cone socket and also the localizer light swung into position. A 21 candle power automobile lamp is used with black paper covering the ionization chamber to avoid reflections. The lever at the right swings it out of the field during irradiation with a snap action switch to shut off the lamp.

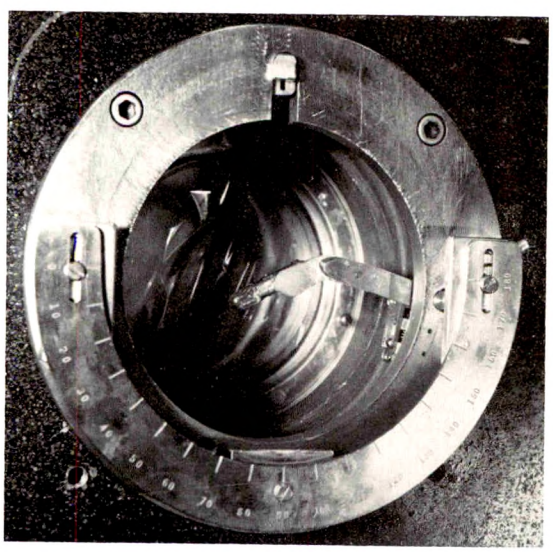


FIG. 8. Cone mounting socket to hold cones on betatron. Light localizer is shown in position for projecting shadow of cross hairs in center of cone onto patient.

PUMPING SYSTEM

Sealed-off electron beam and also dual electron and roentgen ray donuts have been developed by the manufacturer and a sealed-off dual donut is ordinarily used. In order to have immediate replacement capability and to provide for experimental purposes, a continuously pumped donut was also assembled. Apiezon "W" and Araldite An-100 were used for sealing. A VMF-260 diffusion pump using Dow Corning-260 silicone oil reduces the internal pressure to less than 1×10^{-6} mm. of Hg. This pump was suspended on a rotating high vacuum joint, enabling the betatron to rotate as usual (Fig. 9). Figure 10 shows the constructional details of the joint. The lubricant on the "O" rings was Dow Corning high vacuum grease. Fore pump evacuation between the rings prevents any bubbling into the high vacuum side during

rotation. The injector filament was protected by a circuit which incorporated a Phillips-type vacuum gauge.

ELECTRON OUTPUT

Electron beam currents of the order of 10^{-8} amperes are obtained at present. After scattering, the output at the end of the patient treatment collimator at various energies is 50 to 150 rads/min. in water as measured by a calibrated thimble chamber. The output may be limited either by reducing the injector filament current, or by reducing the injector voltage. Only the latter method is safe with sealed-off donuts with thoriated filaments. Either method may be used with pure tungsten injectors, but the former method is preferred to increase filament life. Historically, output has often been expressed in terms of r which has been defined as the reading of a 25 r Victoreen thimble chamber at a depth of 1.5 cm. in a polystyrene block. The N.B.S. calibration factor obtained for 250 kvp. roentgen rays (2.0 mm. Cu half-value layer) is applied. The r as defined corresponds closely to 1 esu./cc. in an air cavity in polystyrene. The ratio of absorbed dose at equilibrium depth in water to the r varies from about unity

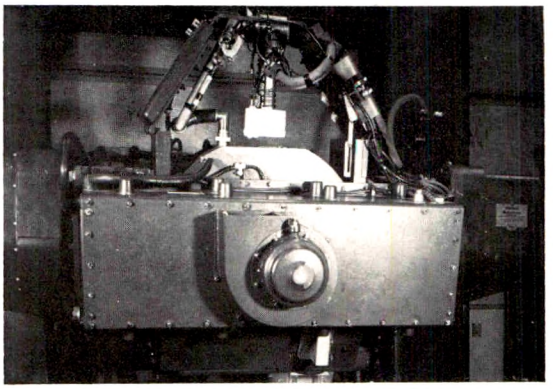


Fig. 9. Pumping system suspended on rear of betatron.

below a few mev. down to about 0.85 for 25 mev. electrons. Ferrous sulfate (Fricke) dosimeter calibrations indicate that over the depth of the electron beam in water an average value of 0.80 rads/r may be used with adequate accuracy in the energy range from 10 mev. to 25 mev.

DOSE DISTRIBUTIONS

The relative distribution of cavity ionization produced by electrons in a water phantom was measured with a 4 mm. diameter air cavity chamber and an automatic isodose recorder. The recorder consisted of

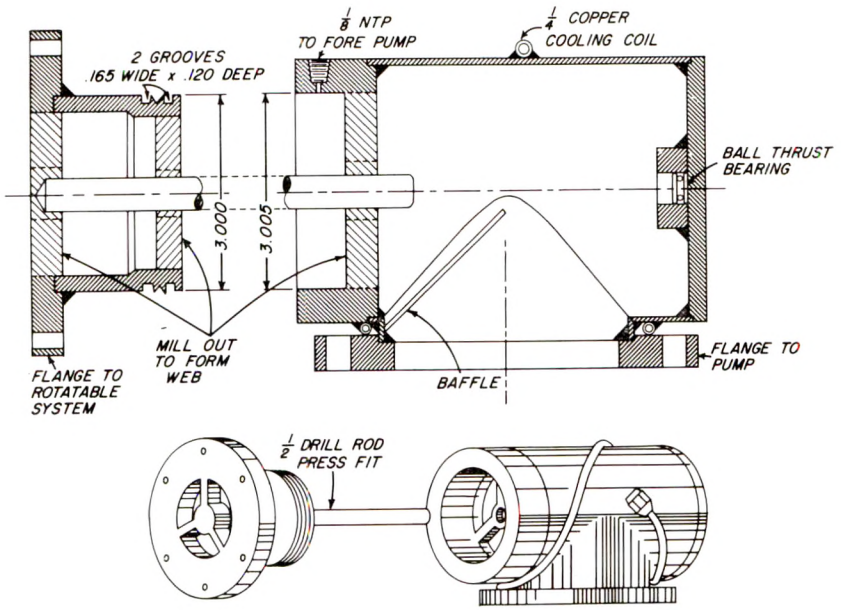


Fig. 10. Construction details of rotating seal.

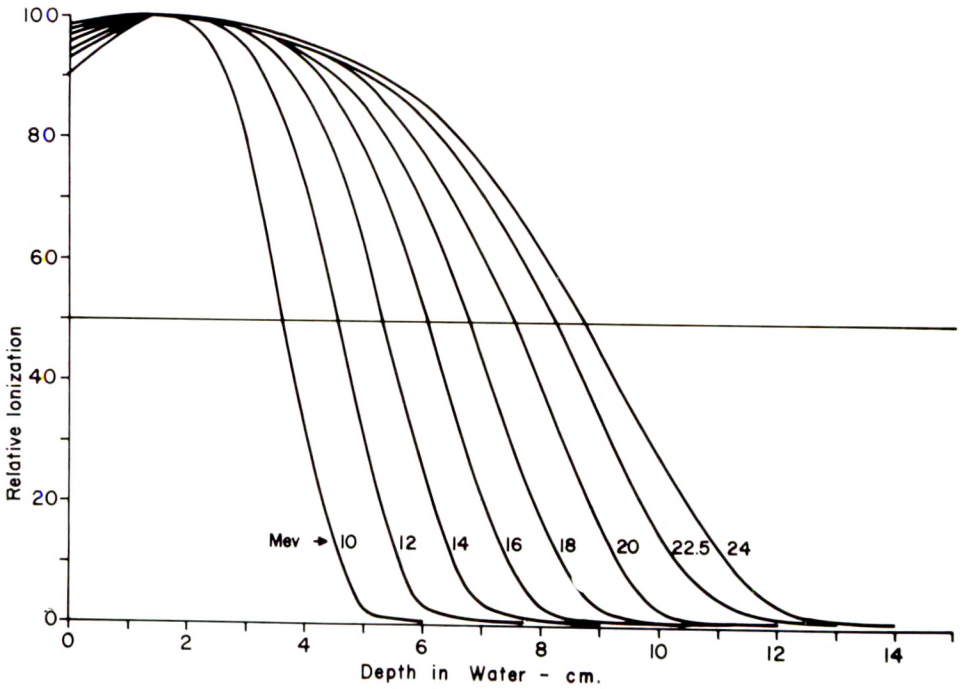


Fig. 11. Central axis depth dose of electron beam; energy range 10–24 mev. (scatterer 0.01 inch Pb); field size 6×8 cm.

4 units: a dual channel D.C. amplifier, a self-balancing ratio unit, a servo mechanism for controlling the position of a probe detector (small ionization chamber in the water phantom), and an automatic plotter which recorded the position of the chamber in any plane. The details of this instrument have been previously described.[2] Before plotting the complete cavity ionization distribution in any plane, the distribution along the central axis of the beam was measured; this procedure was used to check the isodose curves obtained with the automatic plotter.

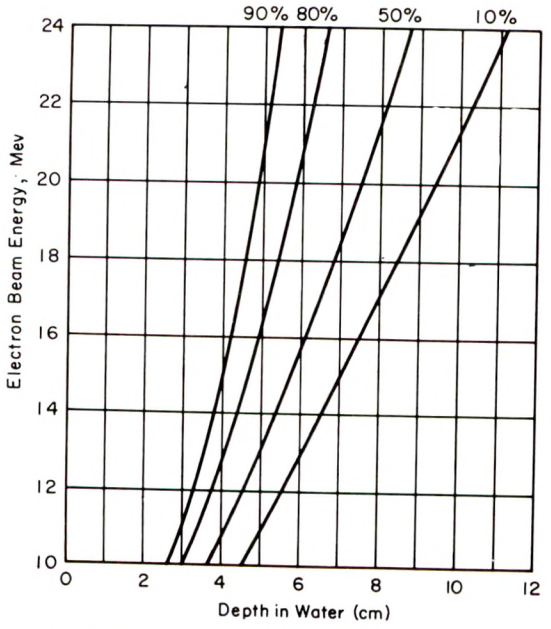


Fig. 12. Variation of percentage depth dose with electron beam energy; energy range 10 to 24 mev.; field size 6×8 cm.

The main factors which influence the isodose distributions include the energy of the incident electrons, the material and thickness of the scatterer between the donut window and the irradiated surface; the design of the collimator and its field size; the orientation of the plane in which the ionization distribution is measured in relation to the donut plane (parallel or perpendicular); and the angle of incidence of the electron beam. The number of possible arrangements is greater than when a roentgen ray beam is used. Representative isodose distributions for normal incidence are shown in Figures 13–19. A detailed

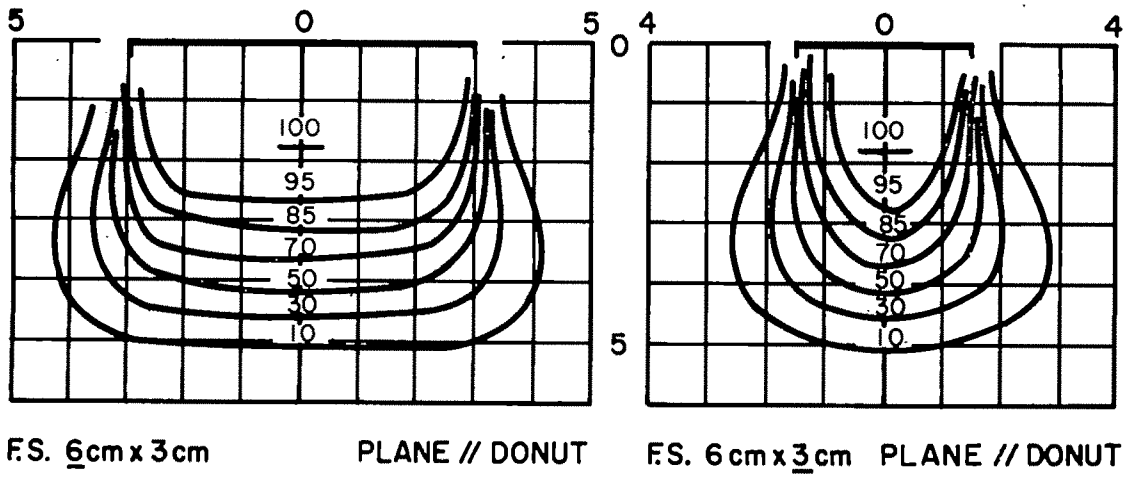


Fig. 13. Electron beam isodose curves; 10.4 mev.

study of the influence of such factors as scattering oil material and collimator material on depth dose distribution will be published subsequently.

CENTRAL AXIS DEPTH DOSE VS. ENERGY

Measurements of relative cavity ionization density along the central axis for field size 6 cm.×8 cm. were made for every 1 mev. interval from 10 mev. to 20 mev., and at 22.5 mev. and 24 mev. For other field sizes the cavity ionization density was measured at several energy intervals. The effect of changing the energy of the incident electrons on the relative cavity ionization along the central axis is best illustrated in Figure 11. The curves show that the relative cavity ionization at the terminal depth drops sharply at lower energies and is less abrupt at higher energies. These results are similar to those reported earlier.[9] The relationship between the depth dose distributions and the ranges of the electrons has been previously discussed.[4] In Figure 12, the depths at which relative cavity ionization of 90 per cent, 80 per cent, 50 per cent, and 10 per cent occurs are plotted against energies from 10 mev. to 24 mev. The gain in depth by changing from a low energy to a high energy, at a high percentage relative ionization, is less than that for low percentage relative ionization. For instance, Figure 12 shows that, as the energy is varied from 10 mev. to 24 mev.,

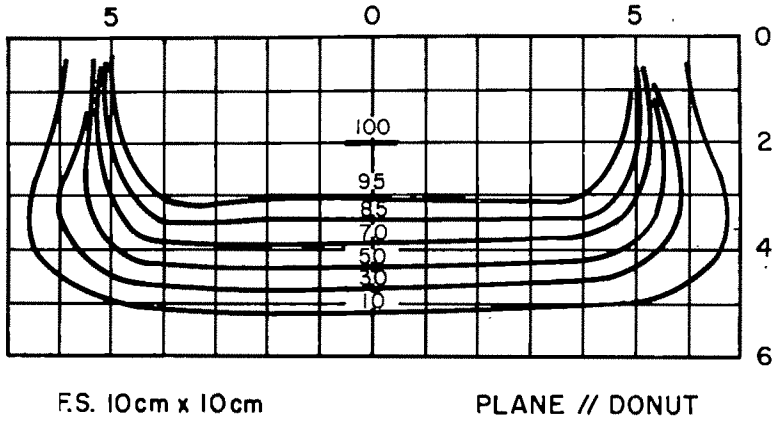


Fig. 14. Electron beam isodose curves; 10.4 mev. (scatterer 0.02 inch Al).

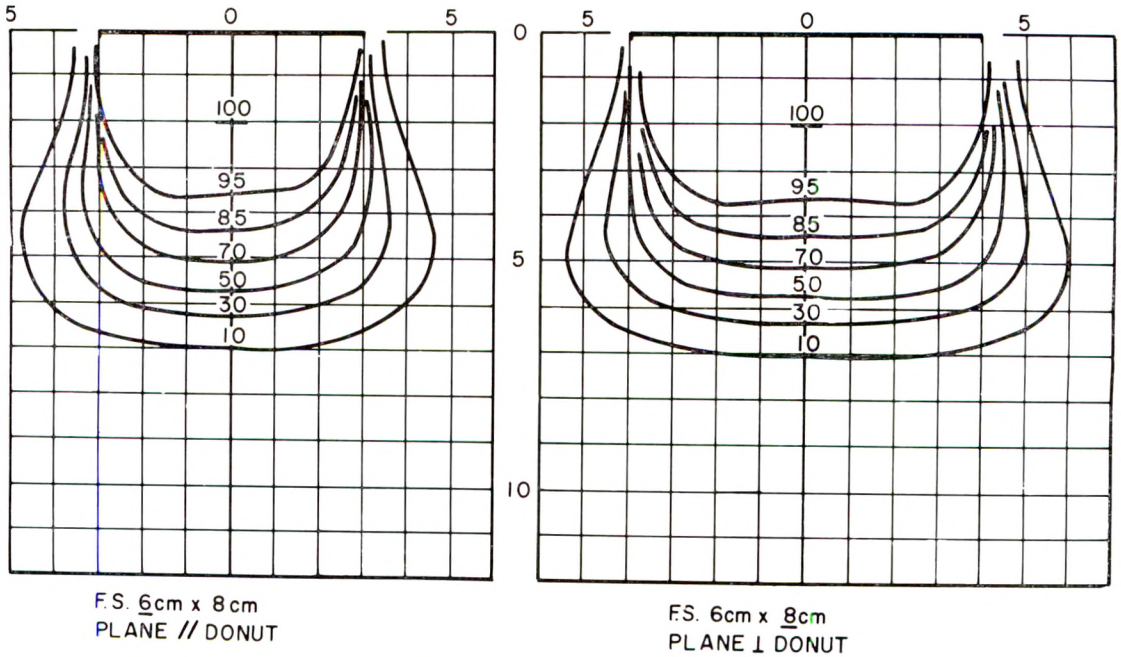


FIG. 15. Electron beam isodose curves; 15 mev. (scatterer 0.01 inch Pb).

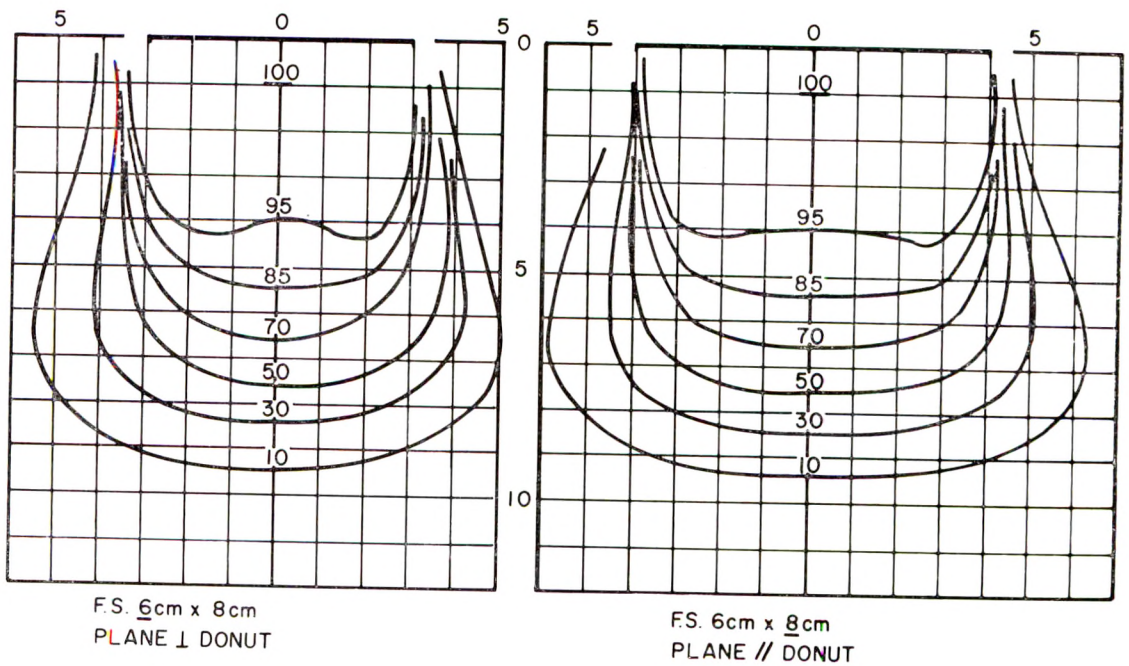


FIG. 16. Electron beam isodose curves; 20 mev. (scatterer 0.01 inch Pb).

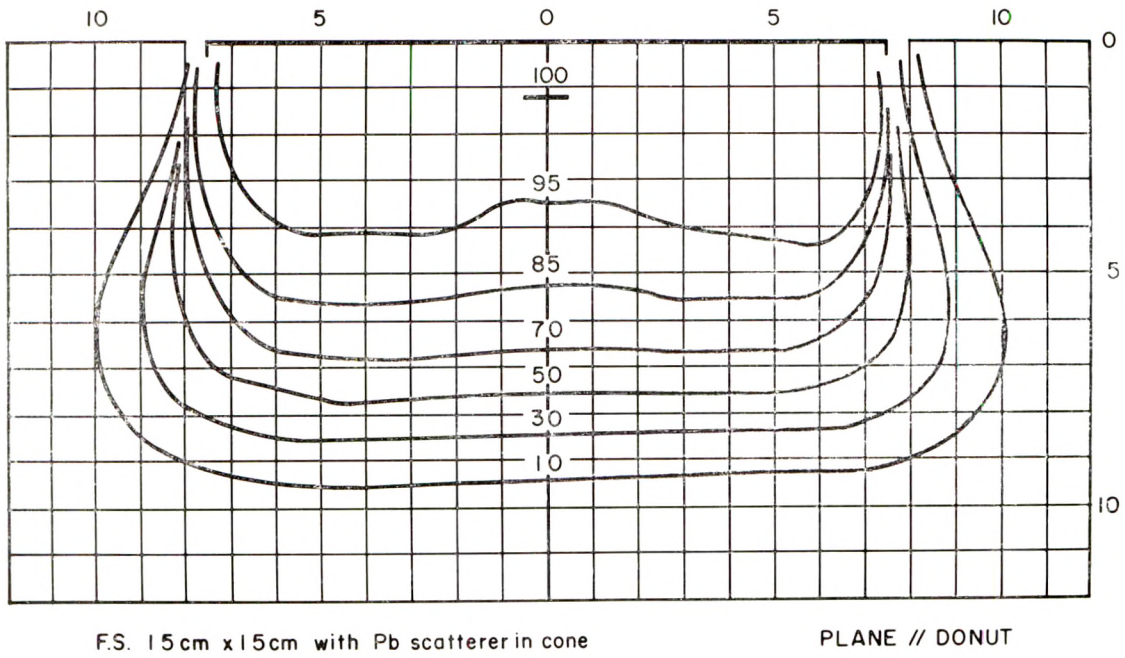


FIG. 17. Electron beam isodose curves; 20 mev. (scatterer 0.01 inch Pb).

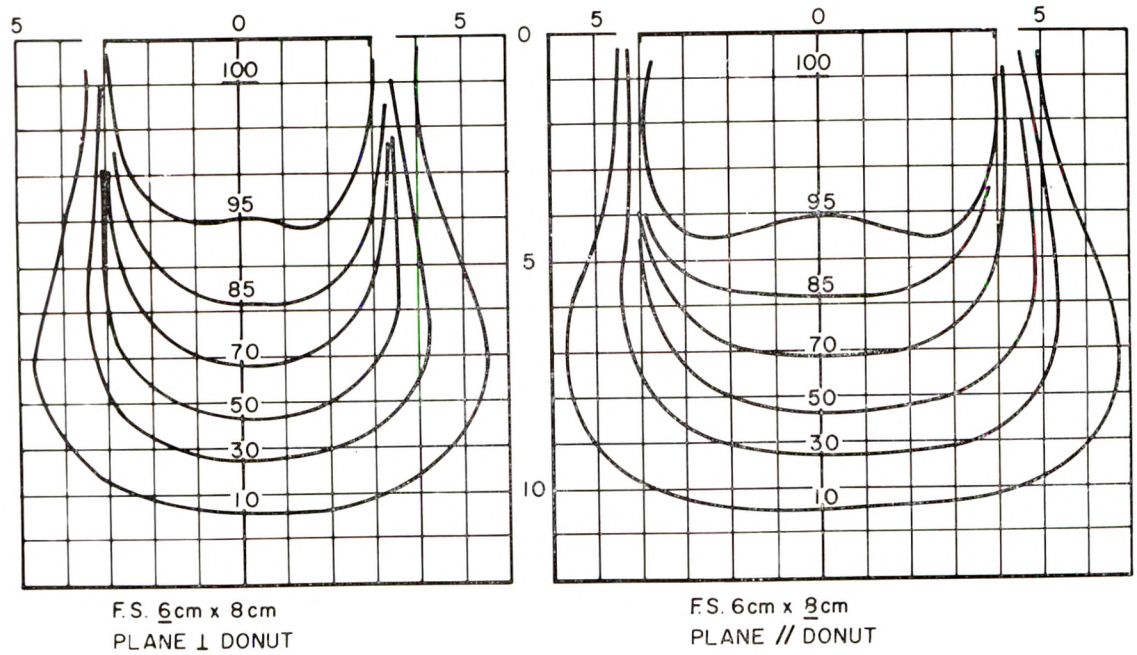


FIG. 18. Electron beam isodose curves; 22.5 mev. (scatterer 0.01 inch Pb).

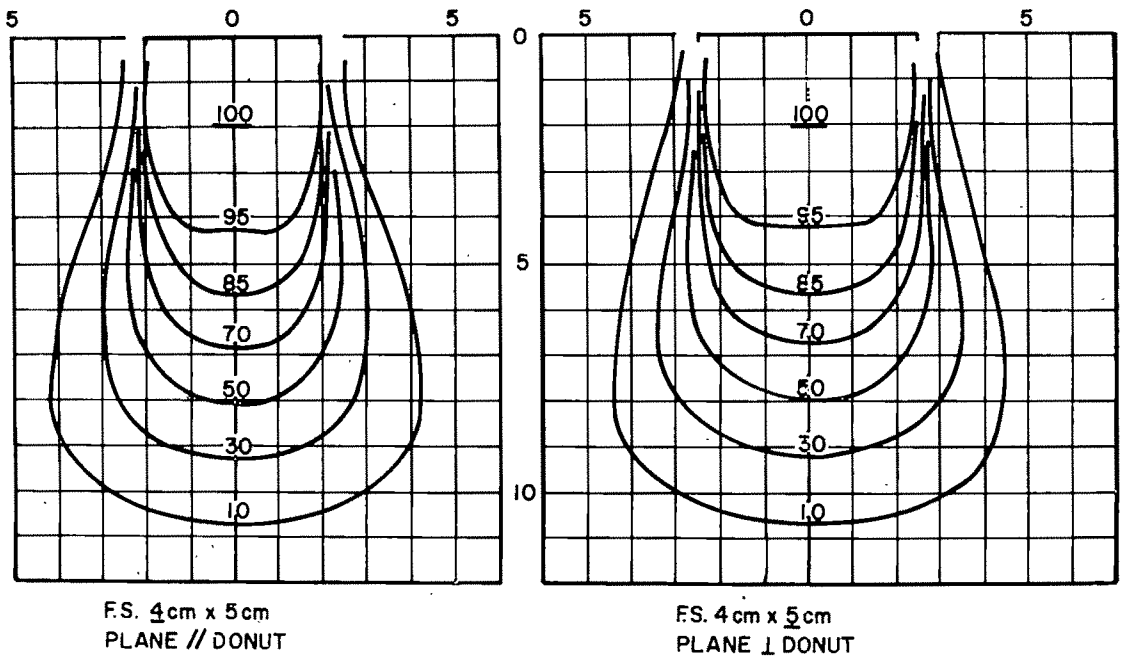


FIG. 19. Electron beam isodose curves; 24 mev. (scatterer 0.01 inch Pb).

the increase of depth for 90 per cent relative ionization is only about 2.6 cm. and the increase for 10 per cent relative cavity ionization is 6.6 cm.

CENTRAL AXIS DEPTH DOSE VS. FIELD SIZE

The relative cavity ionizations along the central axis for field sizes 15 cm.×15 cm., 10 cm.×10 cm., 6 cm.×8 cm., and 3 cm.×6 cm. were compared. It was found that the effect of the field size on the relative cavity ionization varies with the depth in the water and with the energy of the incident electrons. At higher energies, the variation is more prominent than at lower energies, especially in the lower portion of the curves. For square fields of large size and high energies of incident electrons, it was noted that the dose distributions in the plane of the donut and in the plane perpendicular to the plane of the donut are slightly different. The dose distribution in the plane of the donut is more uniform than the dose distribution in the plane perpendicular to the donut. Therefore, the dose distribution in the plane perpendicular to the beam axis for a square field is slightly rectangular. This phenomenon is to be expected since the electrons are channeled into a rectangular cross section by the geometry of the rectangular channel in the magnetic shunt. For small field sizes and lower energies, this effect is not perceptible.

ISODOSE DISTRIBUTION VS. ANGLE OF INCIDENCE

The isodose distributions were also measured for various angles of incidence of the electrons on the water (10, 15 and 30 degrees), and one of these is illustrated in Figure 20. It was noted that the central portion of the isodose curves remained parallel to the water surface for oblique angles of incidence. This is different from isodose distribution produced by roentgen rays for oblique irradiation in which the curves near the center of the field are inclined at an angle approximately half way between the plane perpendicular to the axis and a plane parallel to the water surface.[10]

ISODOSE DISTRIBUTION WITH POLYSTYRENE WEDGES

In the use of roentgen rays, wedge absorbers are necessary in instances where the body surface is irregular or inclined with respect to the axis of the beam. Similarly, with electrons wedge shaped absorbers are useful in altering the dose distribution beyond the body surface. A significant difference exists in that since the electrons are absorbed relatively uniformly with depth (approximately 2 mev. loss per 1 cm. of water), the shift of the isodose distribution in the body beyond the wedge will equal almost exactly the thickness of the wedge. Polystyrene was employed since it is of low atomic number, is firm and easy to machine, and has a density (1.05 gm./cm.^{3}) close to that of water. Bolus material and tissue equivalent wax can also be used. One of the isodose distributions obtained with a polystyrene wedge is shown in Figure 21.

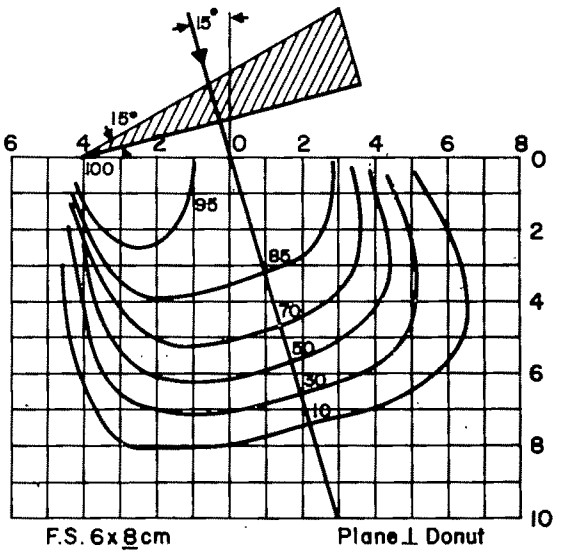


Fig. 21. Electron beam isodose curves; 20 mev. (scatterer 0.01 inch Pb); beam 15° from vertical incidence, with polystyrene wedge.

MULTIPLE FIELD COMBINATION

To date, a combination of more than two fields seldom has been used in electron beam therapy. The 50 per cent dose for the 24 mev. electrons is at a depth of 8.7 cm. and for 10 mev. electrons is at a depth of 3.6 cm. The contribution of dose at the center due to any field in a multiple field combination deeper than 8.5 cm. is too small to be of much practical value. If a single portal is employed, the useful depth is much less; at 6 cm. the depth dose is 85 per cent for a 24 mev. electron beam. The application of the electron beam in therapy is most suitable for lesions near the surface and for those located in regions of small cross section of the patient's body, *i.e.*, diameters not more than 17 cm. With a single field, or two opposing fields, and adjustment of the energy and exposure ratio of the two fields, the clinical requirements can generally be met. Figures 22 through 27 show the general patterns of dose distributions produced with different energies and weighted exposure combinations.

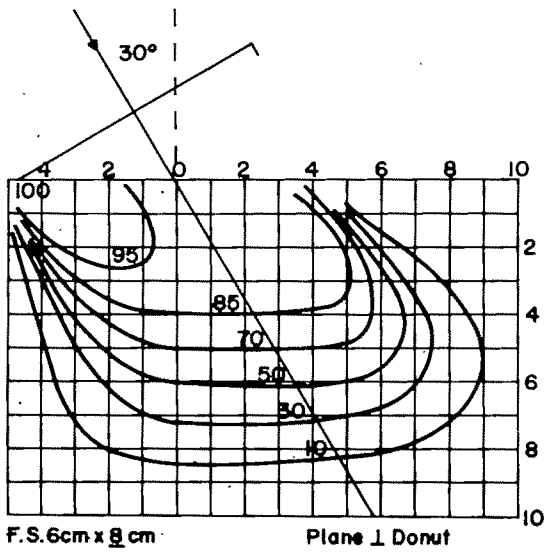


Fig. 20. Electron beam isodose curves; 20 mev. (scatterer 0.01 inch Pb); beam 30° from vertical incidence.

The dose distribution pattern produced by two opposing electron beams of any given field size depends upon (1) the separation of the two fields, (2) the energy of incident electrons to each field, and (3) the exposure ratio between these two fields.

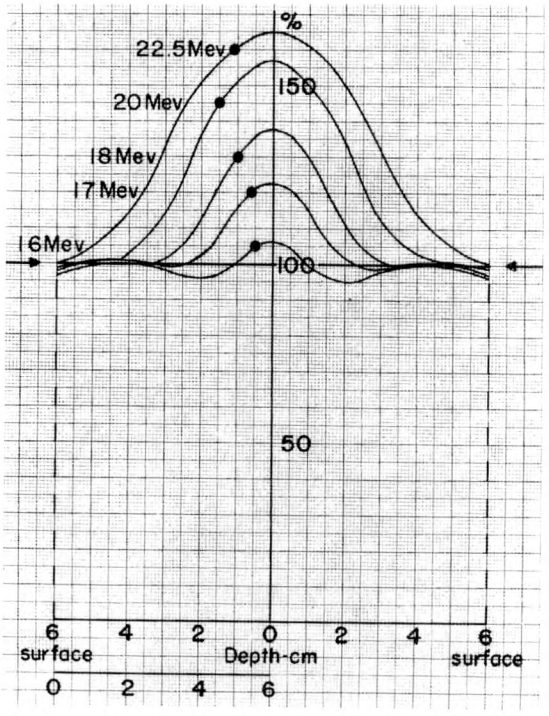


FIG. 22. Central axis depth dose of electron beam. Two opposing fields 6×8 cm.; separation 12 cm.; exposure ratio 1:1.

Figures 22 and 23 show the combined central axis depth dose curves plotted for various energies with an exposure ratio 1:1 for separations of 12 cm. and 16 cm., respectively. Figure 24 illustrates the case of a separation of 12 cm. with an exposure ratio 2:1.

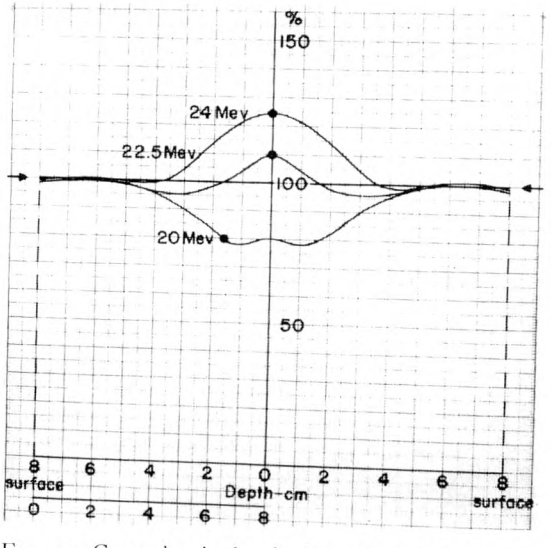


FIG. 23. Central axis depth dose of electron beam. Two opposing fields 6×8 cm.; separation 16 cm.; exposure ratio 1:1.

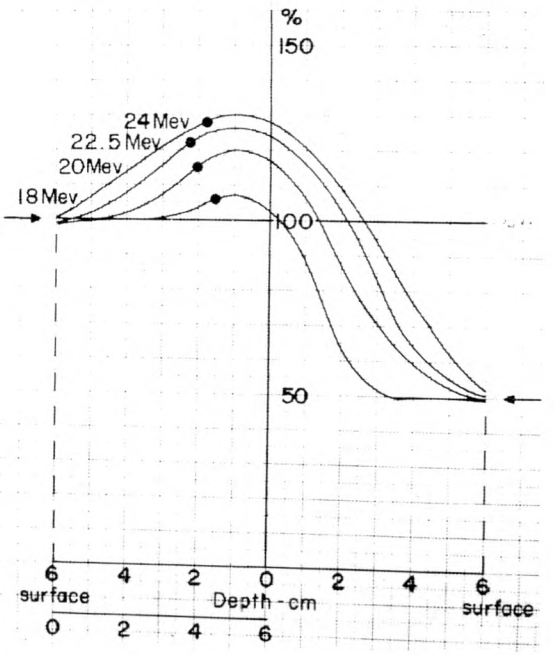


FIG. 24. Central axis depth dose of electron beam. Two opposing fields 6×8 cm.; separation 12 cm.; exposure ratio 2:1.

In the case of equal exposures with both fields, the higher the energy of the electrons employed, the greater is the increase of the dose obtained in the central region. For a given separation there is an optimal energy at which the variation of dose along the central axis is a minimum. Figures 25, 26,

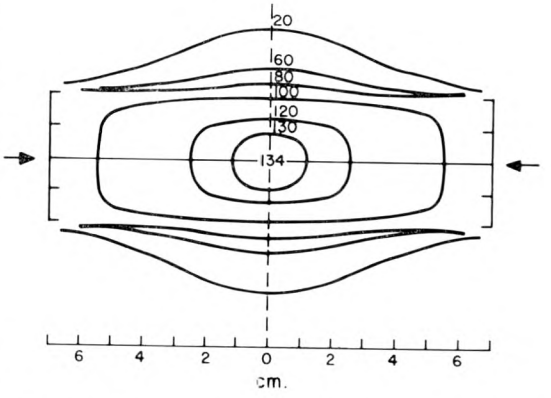


FIG. 25. Dose distribution of two opposing fields; 24 mev.; 4×5 cm.; separation 14 cm.

and 27 are isodose distributions for various separations and relative exposures. Figure 27 illustrates the combination of two fields obliquely. The combination of different energies is also frequently employed in order to manipulate isodose surfaces.

The dose distributions in these figures illustrate some of the dose configurations available by various combinations of energy, field size, field separations, exposure ratio, and angle of approach. Applications of these developments and procedures to cases which illustrate the use of these different techniques are considered in the article which follows.

SUMMARY

This report has described the modification of a betatron for electron beam therapy. Topics discussed have included circuits necessary for orbit contraction, beam monitoring, and energy control. The definition of fields with aluminum and lucite collimators has been considered. Typical dose distributions produced as a function of energy, field size, angle of incidence, and wedging have been presented. The combinations of different beams to obtain various resultant dose distributions have also been described.

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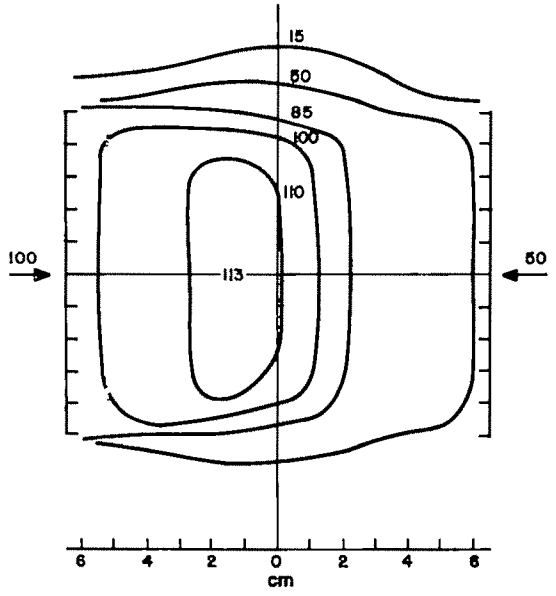


Fig. 26. Dose distribution of two opposing fields; 22.5 mev.; 10×10 cm.; separation 13 cm.; exposure ratio 2:1.

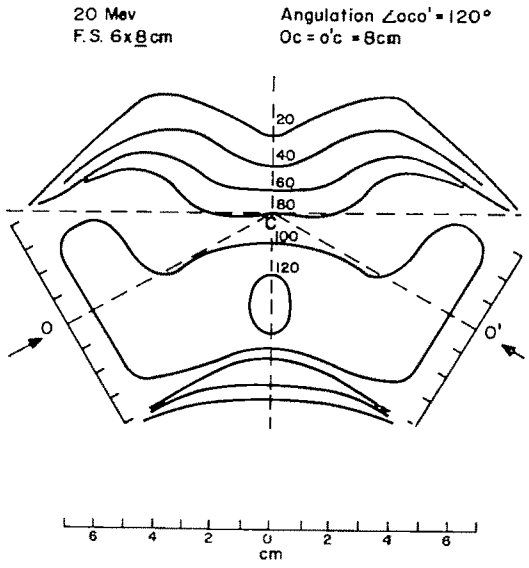


Fig. 27. Isodose distribution for two oblique 20 mev. fields.

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TREATMENT PLANNING IN THERAPEUTIC APPLICATION OF HIGH ENERGY ELECTRONS TO HEAD AND NECK CASES*

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THE physical characteristics of high energy electron beams have made possible some simplifications in the planning and execution of radiation therapy of head and neck neoplasms. A summary of some of the advantages and disadvantages of the use of this type of ionizing radiation is repeated here:[1,4]

1. The surface or entrance dose is low relative to the maximum dose.
2. The depth of penetration of electron beams is proportional to the energy.
3. The energy of the beam and hence the depth of penetration is easily and rapidly adjustable.
4. The effective range of penetration in tissue is limited by the maximal energy of the electron beam; *i.e.*, for the highest energy of the electron beam produced by our betatron, 24 mev., and a field size of 6×8 cm., the 50 per cent dose is at a depth of 8.7 cm. Practically, this limits the use of any particular field to a skin tumor distance not exceeding 8.7 cm.
5. Because of the depth limitation, the electron beam in this energy range, 6 to 24 mev., can only be used for treatment of regions with a small cross section if tumors near the midline are to be treated.
6. The electrons in this energy range (6 to 24 mev.) in addition to a finite range also show an abrupt decrease in dose. In practice this eliminates any significant exit dose even in regions of the body of small cross section.

The purpose of multiple fixed-field techniques with conventional roentgen ray beams is to obtain a higher dose in the tumor than in the surrounding normal structures. In regions of small cross section, such as the head and neck, the available skin area is limited and there is little choice both in the positioning and number of fields. In the case of high energy electron beams, the patterns of dose distribution and the ability to adjust to energy of the electron beam has made this objective obtainable by the use of a single field or two opposing fields dependent upon tumor size and position. The characteristics of electron beams thus are admirably suited to the irradiation of lesions on or near a surface of the body provided that they do not extend more than 6 to 8 cm. deep to that surface. A reasonably uniform dose distribution can be obtained throughout the tumor, avoiding significant deposition of energy in the adjacent normal tissues, by choosing an energy of the irradiating beam or beams appropriate to the depth of the tumor (See Fig. 11 of preceding article).

SELECTION OF PATIENTS

The general principles guiding selection of patients for betatron irradiation at Memorial Center have not yet been recorded in the literature and it seems worthwhile to discuss them in detail at this time, both for electron and for roentgen ray beams.

Since, with the roentgen ray beam, the departure from conventional energies was considerable, the following criteria were

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established prior to the institution of any patient treatment:

(1) Initially only patients with extensive disease, or types with poor probability of favorable response would be accepted; *i.e.*, treatment with conventional techniques, either surgical or radiologic, not likely to be followed with a good response.

(2) Patients with a general condition such that probable survival after treatment would permit adequate observation of tumor and of normal tissue response.

(3) Patients with tumors which normally advanced by direct extension or regional lymph node metastasis. If the cancer were favorably modified, the patient would accordingly receive full benefit in terms of longevity.

(4) In order to evaluate normal tissue response in the various regions of the body, an attempt would be made to select a tumor from the various major body subdivisions which in addition satisfied the above criteria.

(5) In order to permit accumulation of data in a reasonable time, tumors the incidence of which was high were elected.

(6) With the foregoing in mind it was decided to concentrate initially on patients with the following lesions: (a) epidermoid carcinoma of the base of the tongue, (b) adenocarcinoma of the pancreas, (c) invasive carinoma of the bladder, (d) glioblastoma multiforme of the brain.

Obviously, it would be desirable to randomly assign patients to a conventional method of treatment and to betatron treatment. It was not possible to do so. The lack of such a control is serious and means that the possibility of errors of interpretation is increased. These can occur in two directions. Differences in response may be missed, and a difference attributable to variables other than energy of incident beam may be assigned to that variable. It is hoped that in the future better design of clinical investigative procedures will be possible.

Experience with the response of head and neck lesions was clinically deemed favorable with the roentgen ray beam. When the electron beam became available for clinical trial, it was decided to concentrate major attention on patients with primary lesions of the head and neck for this reason, and also, importantly, because the usual interfield separation is less than 17 cm.

In general, the patients treated have had large primary lesions and in many instances these were combined with bulky regional metastases ($T_{3-4}N_3$). A few cases have presented early lesions. A significant percentage has had previous surgery or radiation therapy, and a few have had both types of treatment. These circumstances influenced the technique and problems of treatment.

Techniques developed in treating head and neck cases are discussed in this paper, including the choice of energy, weighted exposure and unequal energies to two opposing fields, and double exposure with different energies and field sizes.

The examples presented illustrate problems encountered in daily clinical practice and the treatment techniques in actual use.

PLANNING PROCEDURE

The procedure of planning is similar in many respects, to that applicable to other forms of radiation therapy. After the usual data for diagnosis are obtained (history, physical examination, and biopsy) the following steps[2,4] are carried out:

1. Tumor size, location and extent. The methods are those used with other radiotherapy machines and include in addition to the clinical examination, endoscopic procedures, examinations under anesthesia, and roentgenography (including laminagraphy where indicated). Gold seeds may be used for accurate centering of the lesion.

2. Plaster cast. A plaster case is made of a light weight resin-plaster.

3. Localization films. Roentgenograms are made with the patient in the cast to relate the position of the tumor to the bony landmarks, gold seeds when used, and to the fixed points on cast.

4. Graphic representation of the tumor. Contours of the principal plane or planes are drawn with the tumor diagrammatically represented. The normal anatomy is drawn in by the projection of a lantern slide of the anatomical cross section of the body at the same level onto the contour.

5. Treatment technique and prescription. The appropriate number and size of treatment fields are chosen to give the optimum and most uniform dose distribution to the tumor-bearing area. At this time the energy level for each treatment field is determined as well as the frequency of usage of each field.

6. Plaster cast completion. The treatment portals which are drawn on the localization films are then transcribed on the plaster cast and these areas are cut out.

7. Check films. Roentgenograms are obtained utilizing a portable diagnostic unit with the patient in the exact treatment position. The roentgen ray tube is centered to the position of the theoretic exit and the film is positioned in the site of entrance following removal of the treatment cone.

8. Set-up of patient. When all field positions and alignments are correct, the patient is placed in the treatment chair or couch wearing the plaster cast and is immobilized. The area of entrance of the electron beam is demarcated by the light localizer and cross hairs are placed in the treatment cone. The point of exit is determined by a back-pointer. The light-localizer and back-pointer have been described in the preceding article.

The steps followed can be applied for treatment planning in any patient. The major variant is that the energy applied to a given portal may be changed with ease. This permits closer approximation of energy distribution to the volume believed to contain the tumor. The delivery of unequal doses through various portals either with the same energy level to each portal or with differing energies as a further means of approximating the volume of high, uniform dose to the tumor volume has had prior application with photon beams. With the electron beam, it is a variable of more general usefulness.

SINGLE FIELD TECHNIQUE

High energy electron beams in the range of 6 to 24 mev. with low entrance dose, variable depth of penetration proportional to energy, and rapid rate of decrease near the end of the penetration make them an ideal therapeutic tool for lesions on or near the body surface, utilizing a single treatment field. The ease with which the energy level of the beam may be varied can also be used to advantage.

Figure 4 is an example in which a single treatment field was utilized. This was an ideal case for treatment by electron beam. The patient had an epidermoid carcinoma Grade III of the posterior right lateral border of the tongue. The tumor measured 6×3×approximately 2 cm. and was ulcerative and infiltrating. A 3×4 cm. tender, hard, fixed mass of lymph nodes was palpated over the tip of the right mastoid process. The primary lesion was about 3 cm. from the surface and extended to about 4½–5 cm. depth. Electrons at 22.5 mev. with a single field of 6×8 cm. size were used. The tumor received a uniform dose within the 90–100 per cent dose region. The cervical lymph nodes could also be treated by a single field, 4×5 cm., at 12 mev. Radium implantation would have been the ideal form of treatment for the primary tumor, but it was refused by the patient.

Figure 10 illustrates another example of the use of single fields in an instance of an anaplastic epidermoid carcinoma of the nasopharynx metastatic to bilateral cervical lymph nodes. The lymph nodes on the right presented a large, matted, lobulated, fixed mass measuring 11×9.5 cm. The mass extended from the tip of the mastoid process to the clavicle. On the left a 10×8×3 cm. fixed mass extended from the mastoid to the supraclavicular region. In Figure 10 it is to be noted that the energy for each field is different and is regulated by the lymph node thickness. The lymph nodes were treated separately by using a

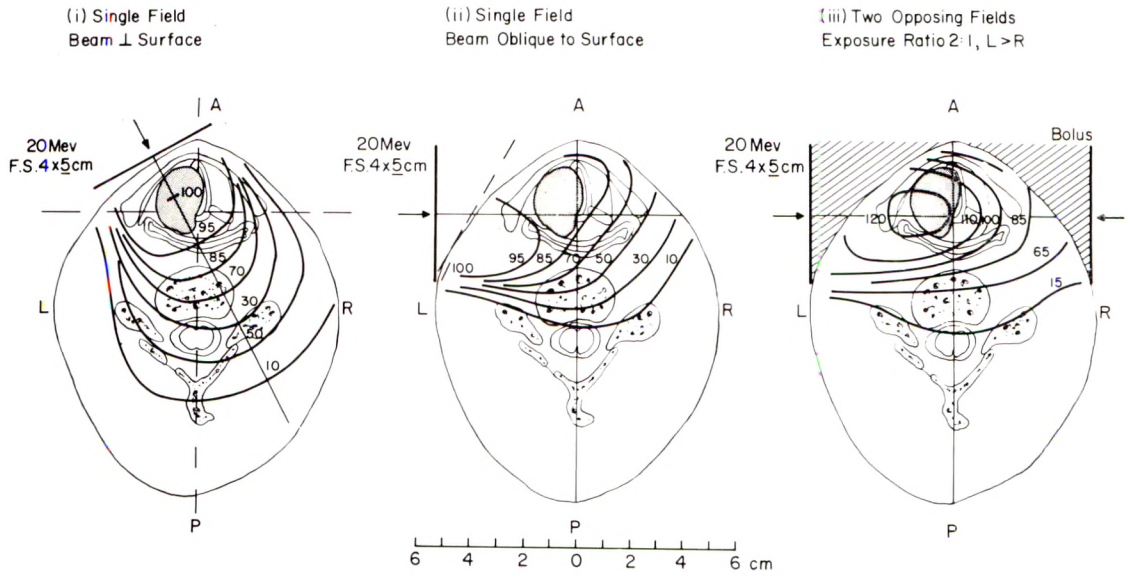


FIG. 1. Patient J.G. Vocal cord. (I) Single field directed perpendicular to the surface; (II) single oblique field without bolus; (III) two opposing fields with bolus.

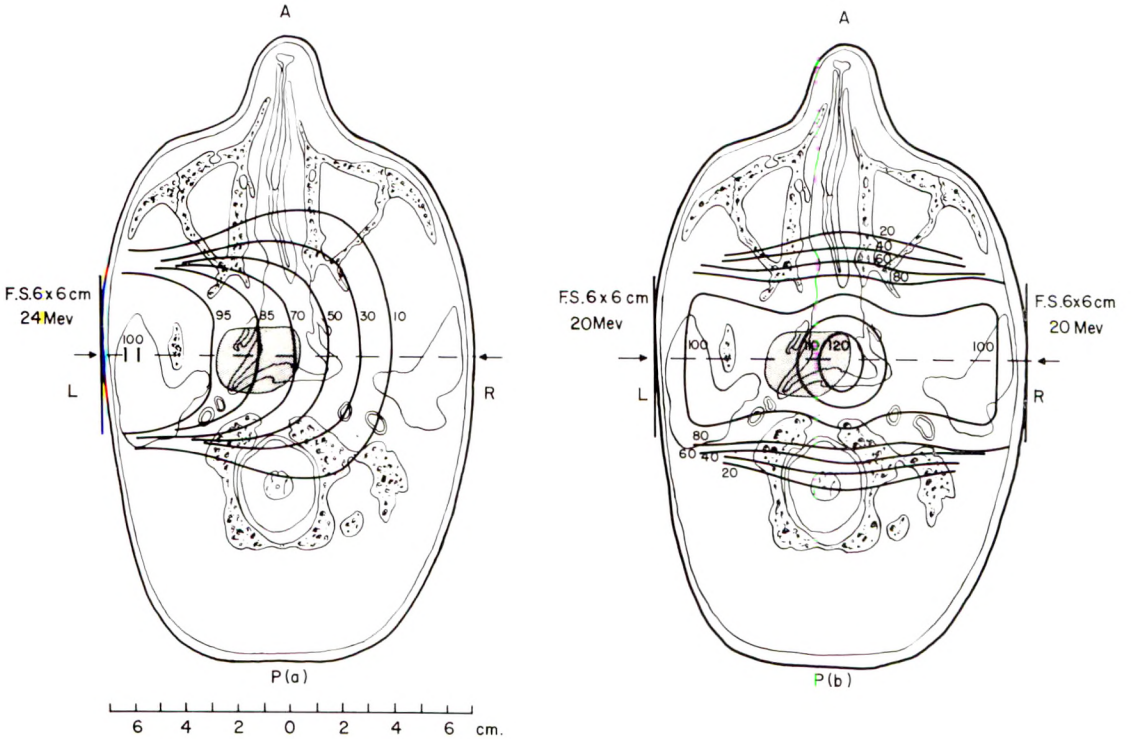


FIG. 2. Patient E.R. Nasopharynx. (a) Single field; (b) two opposing fields with equal energy, exposure ratio 2:1.

12×12 cm. field, 18 mev. for the right cervical lymph nodes, and an 8×10 cm. field, 12 mev. for the left cervical lymph nodes. The dose variation within the lymph node masses was within 10 per cent and very little irradiation was received by the laryngeal and pharyngeal mucosa. In practice, as the masses decreased in size, the energy of each beam was decreased. Thus, a uniform dose was maintained in the required volume and throughout the course of treatment the midline structures received a minimal quantity of irradiation.

Oblique Irradiation and the Use of Wedges

Oblique irradiation occurs when the beam axis is not normal (perpendicular) to the irradiated surface. It may be used in treating patients with neoplasms in the head and neck region when significant changes in surface contour exist, and is a way of maintaining reasonable uniformity of energy deposition throughout the tumor (Fig. 1, II).

This objective may also be accomplished by the use of wedges as discussed in the preceding article. If a polystyrene wedge of the same angulation or curvature as the oblique angle is inserted in the treatment collimator, the isodose curve will be returned to the original position with little decrease of the depth dose. This technique finds utilization in the case of two opposing fields with varying interfield separation to avoid an increased dose in the narrower region of the patient's body (Fig. 1, III).

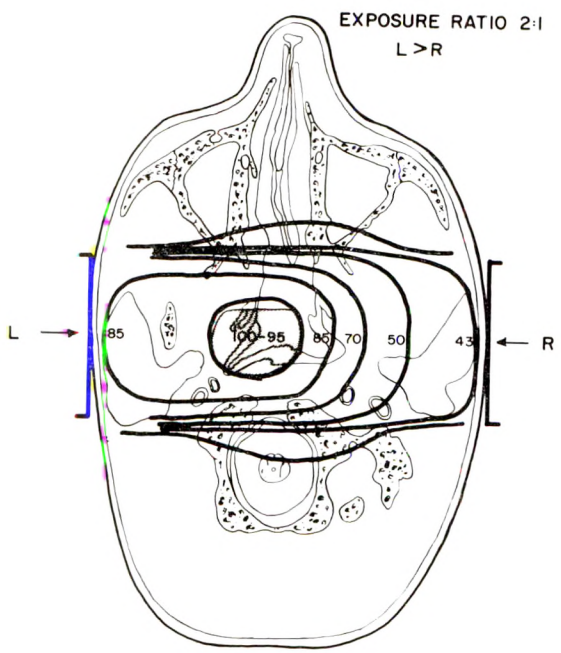


Fig. 3. Patient E.R. Two opposing fields (6×6 cm.), equal energies (24 mev.), exposure ratio 2:1.

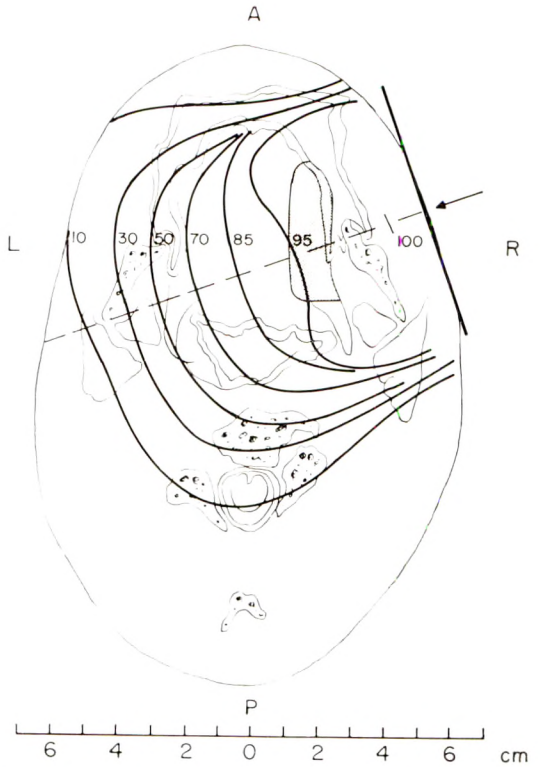


Fig. 4. Patient C.R. Right lateral border of tongue. Single right lateral field (16×8 cm.), 22.5 mev.

Tissue equivalent material such as beeswax or Lincolnshire bolus may also be used to achieve the same objective.

Figure 1 illustrates the principles described above. The patient presented with a postoperative recurrence in the region of the left vocal cord. There was edema of the arytenoids and the ventricular bands which occluded the glottis. On direct laryngoscopy a 2 cm. oval, granular plaque was noted inferior to the arytenoid in the region of the left vocal cord and extended beneath the glottis for 2 to 3 cm.

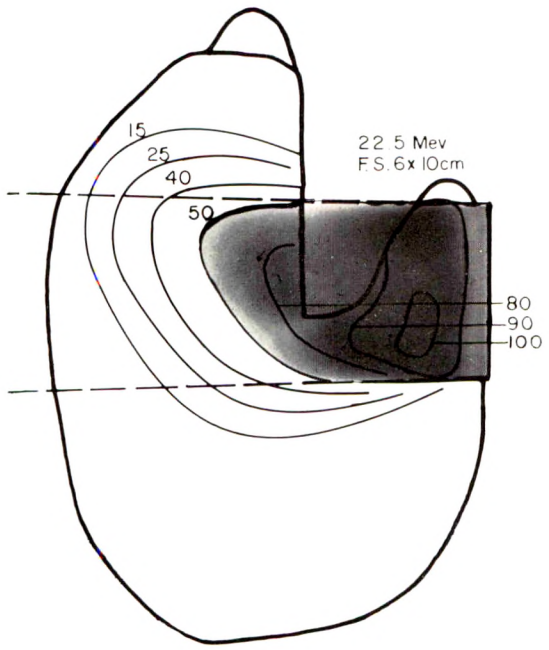


Fig. 5. Patient E.L. Right lateral field.

Figure 1, I presents the use of a single field with the central axis normal to the irradiated surface. The tumor lies between the 95–100 per cent isodose line, but considerable radiation is received by normal structures. In Figure 1, II the beam axis is oblique to the irradiated surface, and the isodose curves are shifted to a position nearly parallel to the contour of the surface. The tumor is within the 85 to 95 per cent dose region and some unnecessary irradiation of normal tissue is avoided. Figure 1, III illustrates the use of bolus and unequal exposure. In this instance the tumor would receive a higher dose than the surrounding normal tissues. However, a greater degree of non-homogeneity is also produced throughout the tumor volume. In addition, some of the normal structures on the right would receive a higher dose than would be received with the other two treatment techniques. The patient was treated with the technique illustrated in Figure 1, II as representing the best compromise for this patient. The weighted exposure technique is discussed below.

Two Opposing Fields and Weighted Dose Technique

Two opposing fields may be used when the lesion or a part of the lesion cannot be treated adequately by a single field provided the lesion is in a suitable region of the body. When the lesion is centrally located or uniform dose distribution is required from surface to surface two opposing fields with equal exposure from both sides should be utilized. The choice of energy depends upon the dimensions of the cross section of the body as shown in Figures 22 and 23 of the preceding article.

Figure 9 illustrates the composite isodose curves utilizing parallel opposing fields and equal energy on each field. The patient in this instance had an anaplastic epidermoid carcinoma of the nasopharynx. On examination, a bulky lesion involved

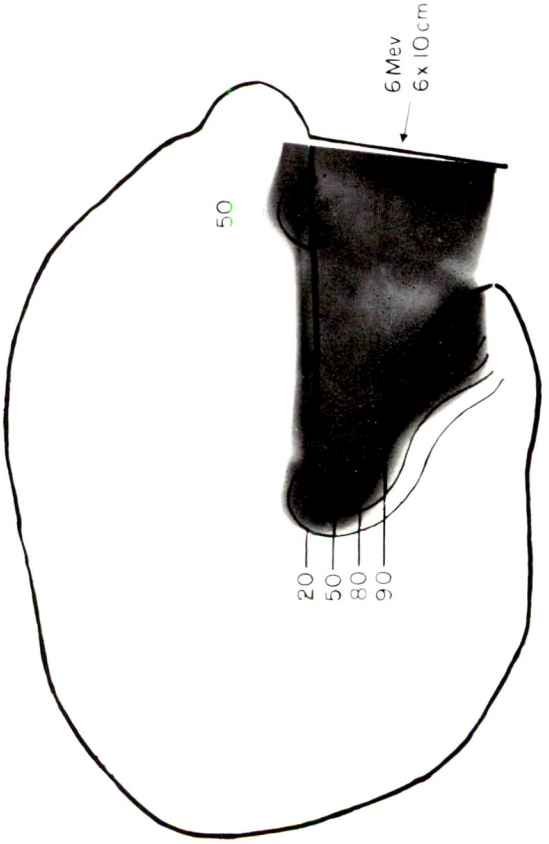


Fig. 6. Patient E.L. Direct anterior field.

the right nasopharynx and protruded into the right choanal area. The tumor also crossed the midline with extension into the left nasopharyngeal vault. Two parallel opposing fields, 4×5 cm., 22.5 mev. with equal exposure were used. Less than 10 per cent variation of dose was obtained throughout the tumor. Treatment of the lymph node metastasis has been discussed under the single field technique.

Differential loading or weighted dose technique results when the exposure for each treatment field for a given lesion is other than equal. The exposure ratio most commonly used is 2:1 for parallel opposing fields. However, the ratio may be 3:1, 3:2 or 4:1, *etc.*, depending upon the treatment requirements in the individual case. In most clinical situations we have found the 2:1 ratio adequate in attempting to produce a homogeneous dose throughout the tumor volume. If an exposure ratio of 2:1 is used, the maximal dose will remain constant from near the surface of greater exposure to the midline. A sharp decrease in dose occurs on the side of lesser exposure. The point of decrease in dose will shift proximal or distal to the midline relative to the side of greatest exposure dependent upon the energy of the electron beams. This technique is suitable for treating lesions off center by 1 or 2 cm., for lesions extending from one surface to the midline. An energy is chosen suitable to the interfield separation, localization and size of the lesion.

The utilization of parallel opposing fields and the weighted dose technique is illustrated in Figures 2 and 3. This patient presented with a recurrent epidermoid carcinoma of the nasopharynx. On examination, the tumor was visualized over the left fossa of Rosenmüller and in the left vault of the nasopharnx. The lesion was eccentrically located but did not cross the midline. No metastatic lymph nodes were detectable in the cervical areas. A single field with the highest available energy, 24 mev., would deliver a dose at 7 cm. depth of 75 per cent. The tumor region would not

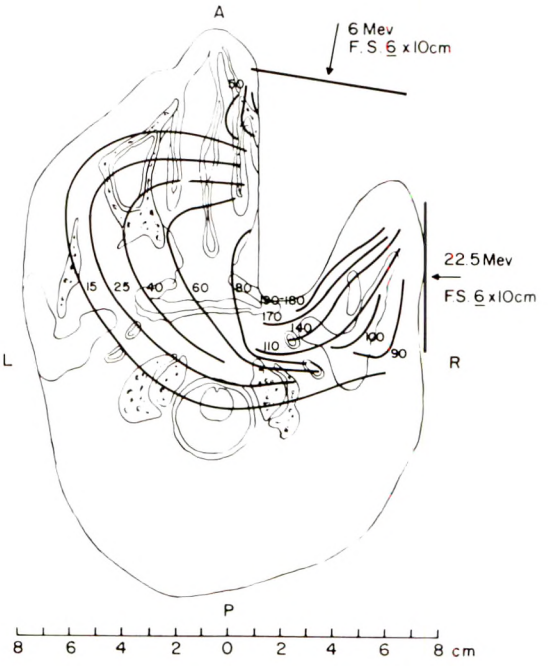


Fig. 7. Patient E.L. Combination of dose distribution. Postoperative recurrent carcinoma of the right buccal mucosa.

receive a homogeneous dose distribution and the subcutaneous tissues would receive more energy than the tumor (Fig. 2, a). Using two parallel opposing fields with a 14 cm. interfield separation, 20 mev. beams, and an exposure ratio of 1:1, a uniform dose would be obtained in the tumor area with, however, a high dose to a large volume of normal tissue surrounding the tumor (Fig. 2, b). For higher energies an increase in dose would be obtained at the central region. There would also be an increase in the dose to the adjacent structures not involved by tumor. The optimal mode of treatment used two opposing fields at 24 mev., with an exposure ratio of 2:1, left greater than right. A uniform dose distribution with a variation of ±2.5 per cent within the tumor was obtained with lower doses to the other tissues in the volume irradiated (Fig. 3).

Two Opposing Fields Using Unequal Energies

When the lesion is eccentrically located

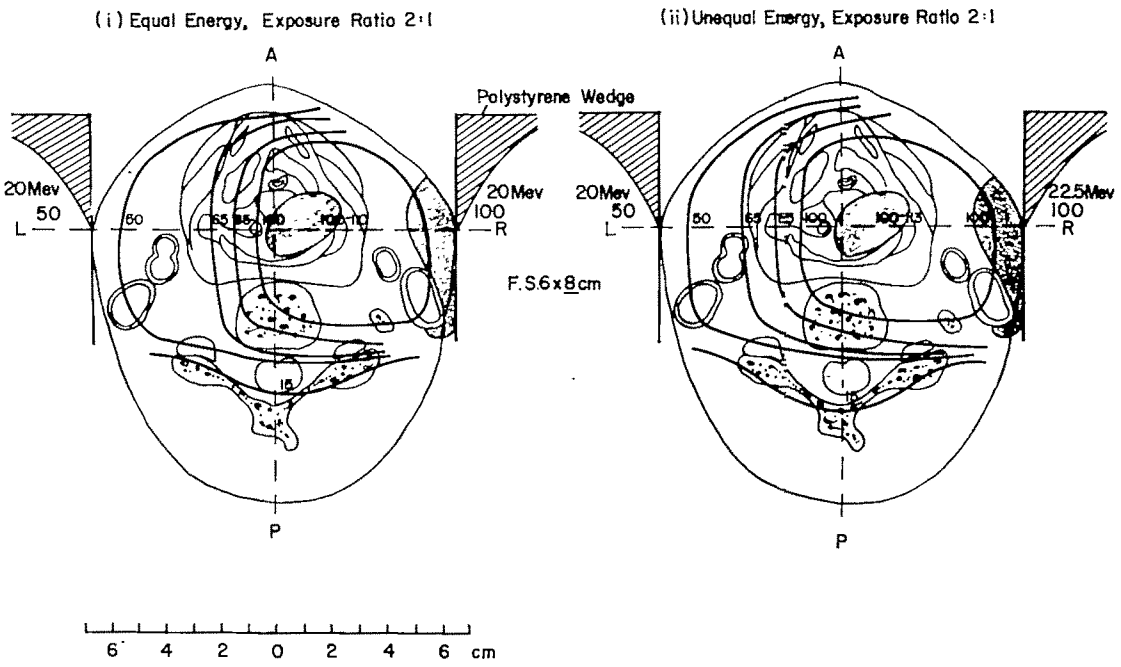


FIG. 8. Patient F.H. External larynx (right arytenoid). (I) Two opposing fields (6×8 cm.) with equal energy, exposure ratio 2:1; (II) two opposing fields (6×8 cm.) with unequal energy, exposure ratio 2:1.

and extends across the midline, the required dose distribution may be obtained by using a beam of higher energy on the side of the bulk of the disease, utilizing in addition a weighted exposure technique with greater exposure on the side of the tumor as described above. This technique results in reasonable uniformity of dose on the side of higher energy and greater dose extending 1–2 cm. across the midline before a sharp fall-off begins.

An example of the technique described above is given in Figure 8, II. This patient had an epidermoid carcinoma, Grade III, of the right arytenoid with right cervical lymph node metastasis. Several treatment plans were studied. (1) A single lateral field was deemed inadequate because the cervical lymph nodes would receive the required dose but the technique would produce a non-uniform dose in the region of the primary tumor. (2) Two opposing fields with an exposure ratio of 2:1, right greater than left, at 20 mev., would give a uniform dose from the surface to the midline (Fig. 8, I). (3) A better dose distribution was obtained with two opposing fields, having an exposure ratio of 2:1, right greater than left. The energy used on the right was 22.5 mev. and on the left 20 mev. This shifted the uniform dose region 1 cm. to the left of the midline resulting in a homogeneous dose to the primary tumor and to the cervical lymph nodes. Polystyrene wedges were used to avoid an area of high dose in the anterior midline region.

TWO OPPOSING FIELDS WITH DOUBLE EXPOSURE ON ONE SIDE USING DIFFERENT ENERGIES

This variant may be used when the lesion is eccentrically located and near the midline, while at the same time there are bilateral lymph node metastases to be treated. A uniform dose from the surface to the midline on the side of the primary lesion may be obtained by using two opposing fields with the weighted exposure technique associated with additional treatments to the side of lower dose with a beam of lower energy. The choice of energy used depends upon the volume to be treated. The addi-

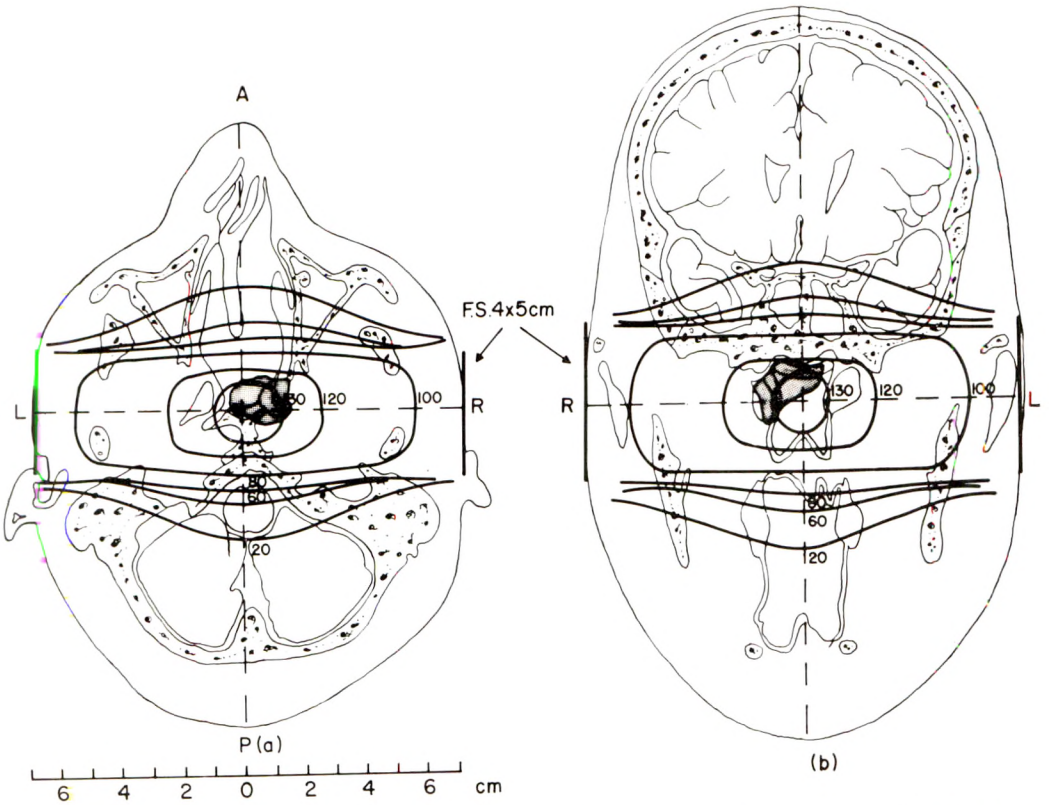


Fig. 9. Patient E.M. Nasopharynx. Two opposing fields (4×5 cm.), 24 mev. (a) Dose distribution to the primary lesion in the transverse plane; (b) dose distribution to the primary lesion in the frontal plane.

tional treatment should be such as to equalize the dose to each site containing cancer.

Figures 11, 12 and 13 represent an example of the treatment technique discussed above. This patient had a squamous cell carcinoma, Grade III of the base of the tongue, metastatic to cervical lymph nodes bilaterally. On examination, there was decreased ability to protrude the tongue. Trismus of the mandible was marked. On palpation, a firm, infiltrating mass was present in the base of tongue and vallecula with extension anteriorly into the body of the tongue on the left. Ulceration of the central portion of the tumor was seen on mirror examination. In the right upper neck, just below the angle of the mandible, there was a 7×6×2 cm. mass which was fixed, firm, and non-tender. On the left, a well-healed 4 cm. scar was present with bronzing and telangiectasia of the skin from previous radiation therapy. Beneath the scar was a firm, partially fixed 4×6 cm. mass. The problem presented was solved in the following manner: (1) two parallel opposing 8×10 cm. fields at 16 mev. with equal exposure of the treatment fields result in uneven dose distribution throughout the tumor (Fig. 11). A greater dose differential in the tumor results if the energy of the beam is increased; (2) the use of two parallel opposing 8×10 cm. fields with exposure ratio of 2:1, left greater than right, energy 20 mev., results in an adequate dose to the left cervical lymph nodes and the primary lesion. A variation of 10 per cent in the dose is thus obtained in the left cervical lymph nodes and the primary neoplasm; (3) the tumor dose delivered to the mass in the right cervical region is increased by the addition of a 6×8 cm. field, energy 10 mev., with the dose the same as given with the right 8×10 cm. field. The 6×8 cm. field lies

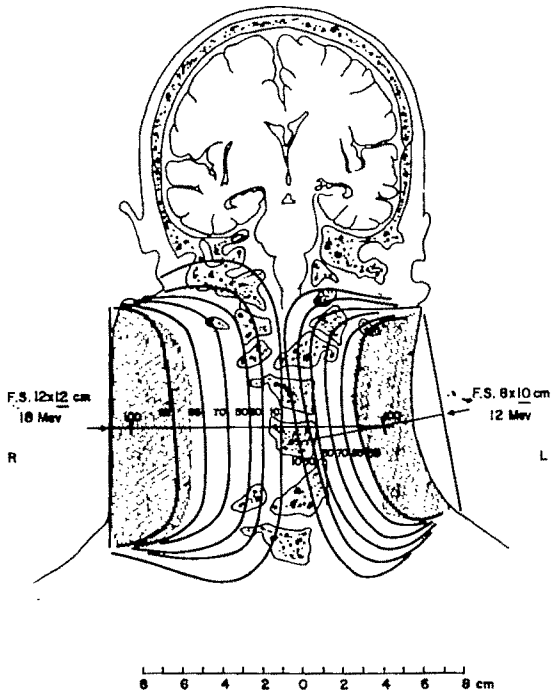


FIG. 10. Patient F.M. Bilateral cervical lymph nodes. Dose distribution to the cervical lymph nodes in the frontal plane.

within the boundaries of the larger field. The total skin dose of this area is equal to that of the left 8×10 cm. field. The right cervical lymph nodes thus receive the desired tumor dose; and (4) the energy level of the 6×8 cm. field may be changed to 6 mev. This gives a uniform dose to the right cervical lymph nodes and decreases the volume of tissue receiving a high dose of irradiation (Fig. 12 and 13).

	Field	Electron Energy Mev	F.S.	Exposure Ratio
I	L	16	10x8	100
	R	16	10x8	100

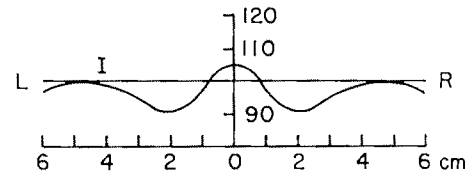


FIG. 11. Patient N.K. Central axis doses of treatment technique I.

MISCELLANEOUS TECHNIQUES

The physical characteristics of the high energy electron beams as previously outlined, in addition of varying the energy level utilized, increase manyfold the number of available treatment techniques utilized with the Allis-Chalmers betatron.

In Figures 5, 6 and 7 the treatment planning of a case is presented in which the flexibility and other physical characteristics of the electron beam are illustrated. This patient presented with a mucinous adenocarcinoma of the right buccal mucosa, recurrent after previous radical surgery. On examination the right orbit and maxillary sinus were surgically absent. There was induration and swelling along the margin of the right cheek, extending onto the mandible. Several tumor nodules were present on the posterior orbital wall, the largest measuring 2×2 cm. Other nodules were present on the anterolateral wall of the soft palate and in the residual upper buccal mucosa. No enlarged cervical lymph nodes were noted. The treatment problem that this case presented was ideal for the use of electron beams. The tumor areas were multiple and were scattered over a

	Field	Electron Energy Mev	F.S.	Exposure Ratio
II	L	20	10x8	100
	R	20	10x8	50
III	L	20	10x8	100
	R	20	10x8	50
	R	6	8x6	50
IV	L	20	10x8	100
	R	20	10x8	50
	R	10	8x6	50

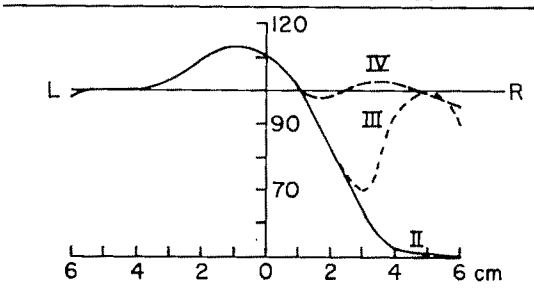


FIG. 12. Patient N.K. Central axis doses of treatment techniques II, III, and IV.

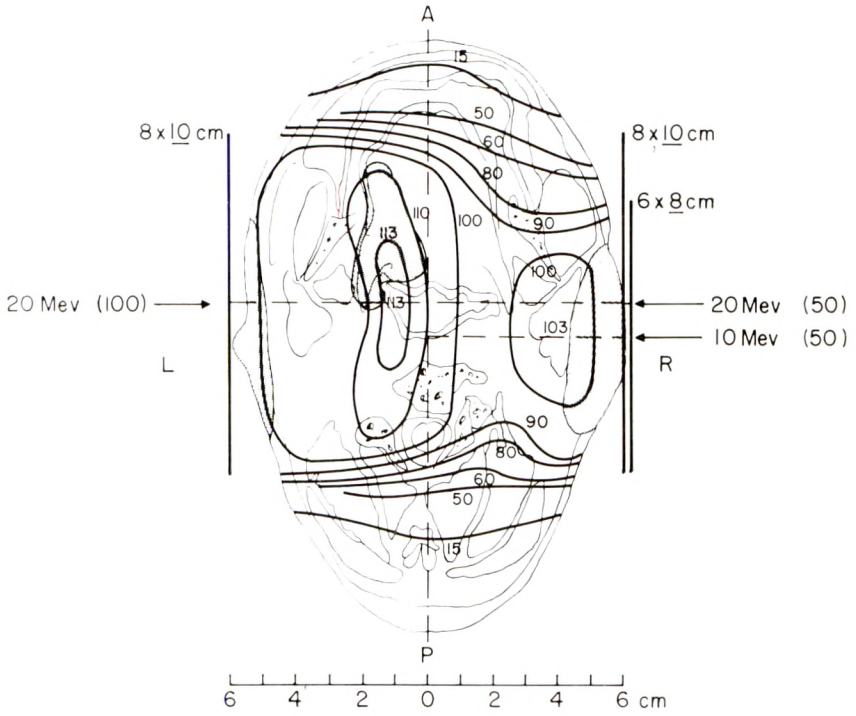


FIG. 13. Patient N.K. Dose distribution of treatment technique IV. Base of tongue with bilateral cervical lymph nodes. Two opposing fields; unequal energy.

large area. The nodules on the posterior orbital wall were adjacent to bone. All of the nodules, however, were near the surface of the operative defect. Two 10×6 cm. fields were used: one a direct right lateral and the other a direct right anterior field. An energy level of 22.5 mev. was employed for the right lateral field and of 6 mev. for the direct right anterior field. A pressdwood phantom[2] was built duplicating the patient's measurements and film measurements were obtained to determine the dose distribution of each field (Fig. 5 and 6). The combined dose distribution is shown in Figure 7.

SUMMARY AND CONCLUSIONS

1. The general principles of treatment of head and neck tumors with high energy, electron beams have been discussed.

2. The philosophy of patient selection for betatron irradiation has been presented.

3. Cases from actual clinical practice have been utilized to illustrate the treatment principles.

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We wish to thank Dr. Jean-Marie Dutreix of Institute Gustave-Roussy for the individual and composite isodose data presented in Figures 5, 6 and 7, while a Fellow in the Memorial Department of Physics.

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BETATRON: ELECTRON BEAM 10–35 MEV.*

CENTRAL DEPTH DOSES AND ISODOSE CURVES

By JACK SPIRA, PH.D., CHARLES BOTSTEIN, M.D., BENSON EISENBERG, M.D., *and* WALTER BERDON, M.D.
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AT THE end of 1961, the installation of a 35 mev. betatron, the Asklepitron 35, was completed. Presented in this report are the central depth dose and isodose charts for the electron beam in the range 10–35 mev. (Table I through V and Fig. 2 through 8.)

The energy of the emergent electron beam was verified using Wideröe's method.[3] Extrapolated ranges of the electrons were determined with depth dose curves in lucite. The electron energy was obtained from the extrapolated ranges by multiplying by the average energy loss of approximately 2 mev. per gm./cm.[2]

The electron beam is defined by a collimator consisting of a diaphragm and a lucite cone. The diaphragm is made of layers of the following metals: first layer, 25 mm. of aluminum to significantly reduce the energies of the electrons with minimum bremsstrahlung; second layer, 8 mm. of iron to absorb completely the electrons; and the third and fourth layers, 5 mm. lead and 4 mm. iron to reduce and absorb brems-

TABLE I

ELECTRON BEAM CENTRAL AXIS DEPTH DOSE DATA

10 mev. Electron Beam; Scatterer 0.3 mm. Cu

Percentile	Depth of Percentile in cm. Water for Various Fields				
	4×4 cm.	4×8 cm.	8×10 cm.	7×14 cm.	14×14 cm.
90	1.9	1.8	2.1	2.0	2.1
80	2.2	2.1	2.5	2.3	2.4
70	2.4	2.3	2.6	2.5	2.6
60	2.6	2.6	2.8	2.7	2.8
50	2.8	2.8	3.0	2.9	3.0
40	3.0	3.0	3.1	3.1	3.1
30	3.1	3.1	3.3	3.3	3.3
20	3.3	3.3	3.5	3.5	3.4
10	3.7	3.5	3.9	3.6	3.7

TABLE II

ELECTRON BEAM CENTRAL AXIS DEPTH DOSE DATA

18 mev. Electron Beam; Scatterer 0.3 mm. Cu

Percentile	Depth of Percentile in cm. Water for Various Fields				
	4×4 cm.	4×8 cm.	8×10 cm.	7×14 cm.	14×14 cm.
90	3.6	3.8	4.0	4.0	3.8
80	4.3	4.8	5.1	5.1	4.8
70	4.8	5.4	5.7	5.5	5.5
60	5.3	5.9	6.1	5.9	6.0
50	5.8	6.2	6.3	6.3	6.4
40	6.2	6.6	6.7	6.7	6.8
30	6.6	6.9	7.0	7.2	7.2
20	7.1	7.4	7.4	7.6	7.7
10	7.9	8.0	8.0	8.1	8.2

TABLE III

ELECTRON BEAM CENTRAL AXIS DEPTH DOSE DATA

24 mev. Electron Beam; Scatterer 0.3 mm. Cu

Percentile	Depth of Percentile in cm. Water for Various Fields				
	4×4 cm.	4×8 cm.	8×10 cm.	7×14 cm.	14×14 cm.
90	4.7	5.1	5.5	5.5	5.7
80	5.6	6.1	7.0	6.9	7.0
70	6.2	6.9	7.8	7.6	7.8
60	7.0	7.7	8.4	8.2	8.5
50	7.8	8.5	9.0	8.8	9.1
40	8.6	9.2	9.6	9.4	9.8
30	9.4	10.0	10.2	10.0	10.5
20	10.4	10.7	11.0	10.7	11.2
10	12.7	—	—	—	—

* From the Department of Radiotherapy, Montefiore Hospital, New York, New York.

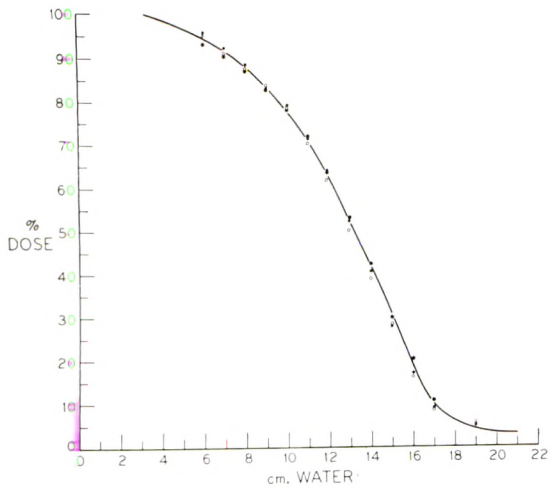


Fig. 1. Central axis depth dose data for electron beam 35 mev., field size 8×10 cm. Curve represents ionization measurements; points were obtained from three independent film measurements.

strahlung originating in the first two layers. This type of diaphragm sharply limits the beam and prevents contamination by secondary roentgen rays and stray electrons. A lucite cone, mounted on the main diaphragm, acts as an additional field limiting device and localizer. Presently available lucite cones define areas of 4×4, 8×4, 10×8, 14×7, and 14×14 cm.

Table IV

Electron Beam Central Axis Depth Dose Data

30 mev. Electron Beam; Scatterer 0.3 mm. Cu

Percentile	Depth of Percentile in cm. Water for Various Fields				
	4×4 cm.	4×8 cm.	8×10 cm.	7×14 cm.	14×14 cm.
90	5.2	5.7	6.9	6.8	7.2
80	6.3	7.0	8.6	8.5	9.2
70	7.3	8.2	9.7	9.7	10.3
60	8.3	9.3	10.7	10.6	11.2
50	9.3	10.3	11.5	11.5	11.8
40	10.3	11.2	12.2	12.2	12.5
30	11.3	12.2	12.8	12.9	13.2
20	12.3	13.1	13.6	13.6	13.8
10	13.8	14.7	14.8	14.9	15.0

A lucite phantom, composed of sheets 30×30×1.25 cm., with a density of 1.18 gm./cm.^{3} was used for depth dose and isodose determinations. Radiation output was

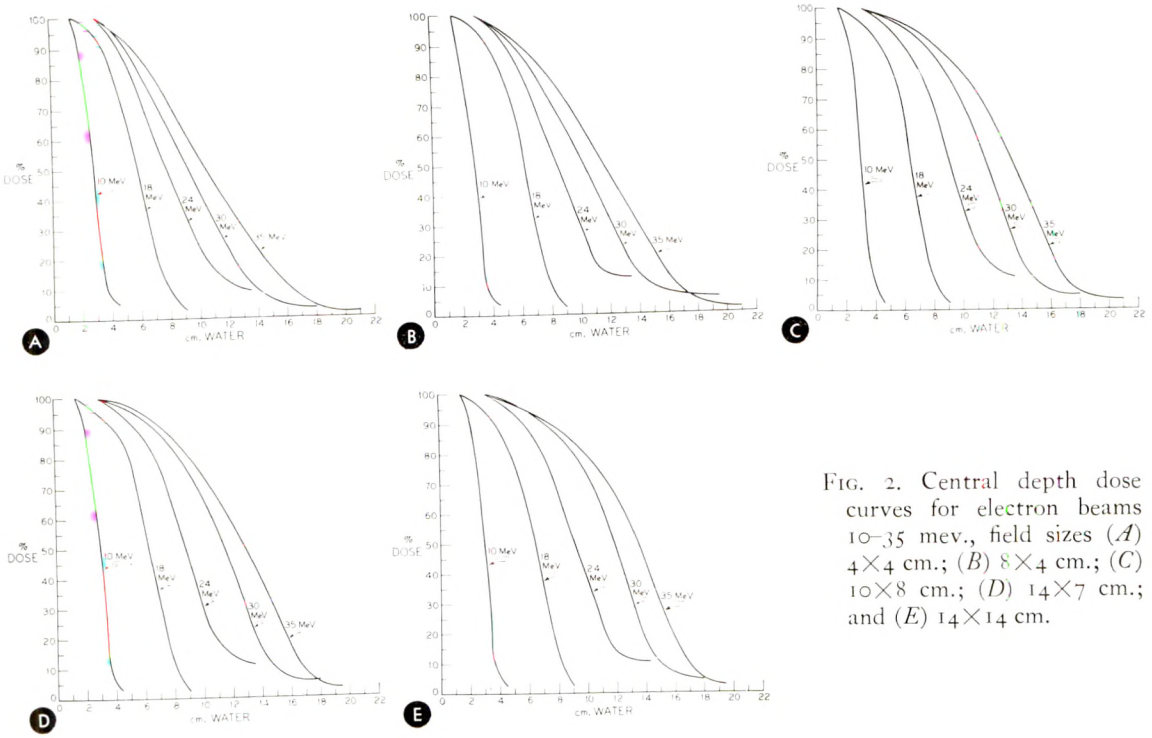


Fig. 2. Central depth dose curves for electron beams 10–35 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

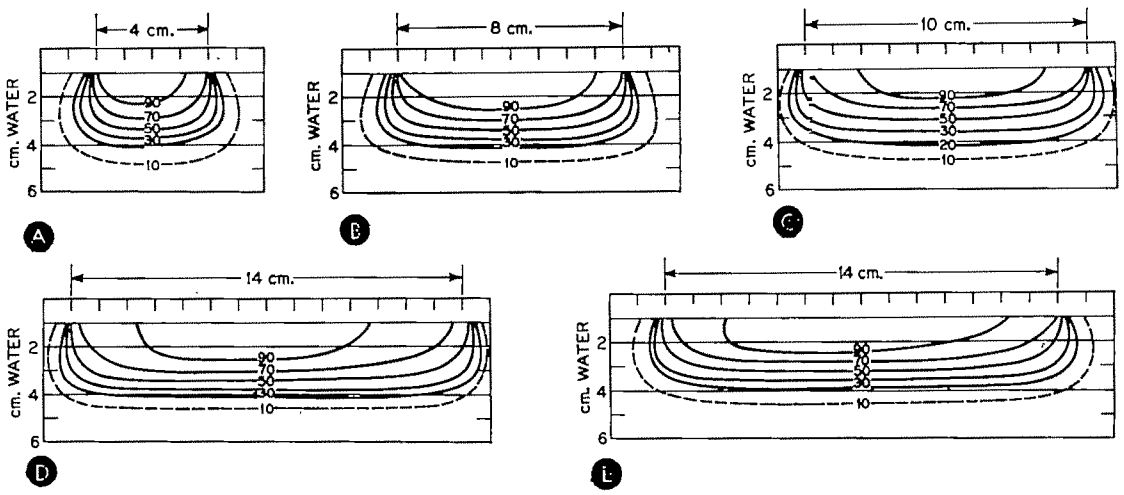


FIG. 3. Isodose charts for electron beam 10 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

measured with a Farmer-Baldwin Mk-2 Sub-standard Dosemeter.[2] This instrument consists of a small thimble ionization chamber (air volume of 6 mm. diameter and 30 mm. length) and an electrometer valve-null indicator, connected by a long flexible cable. The instrument operates on the Townsend balance principle. The ionization current charges a capacitor whose high insulation terminal is connected to the grid of the electrometer valve. As the capacitor becomes charged, the grid potential of the electrometer valve and its anode current will change. At the end of exposure, balance is restored by applying voltage from a potentiometer. The voltage is a measure of a dose readable directly in (betatron) roentgens. The electrometer valve merely serves as a balance indicator, and the accuracy of the instrument depends only on the stability of the ionization chamber, capacitor, and voltmeter. The Farmer-

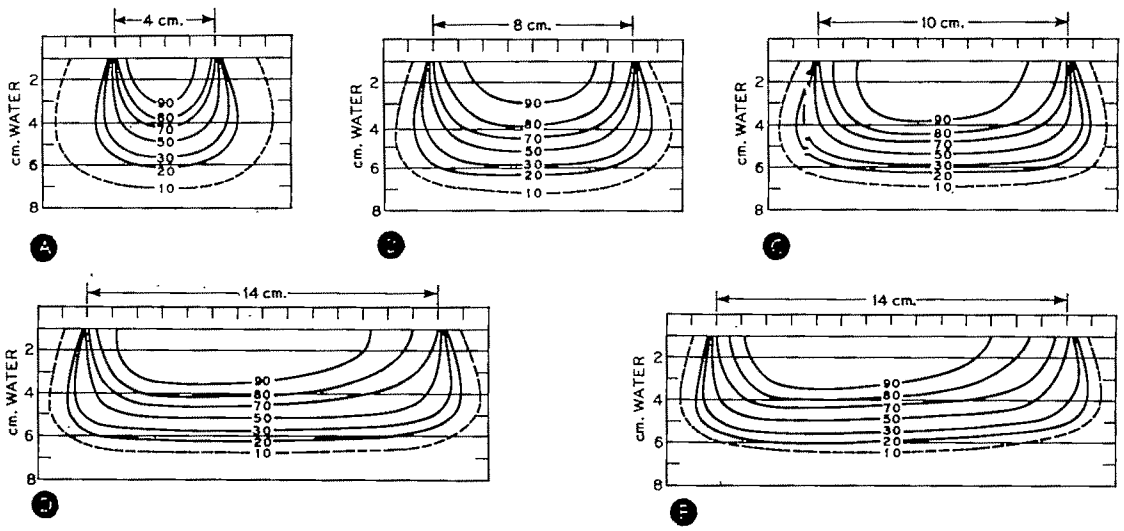


FIG. 4. Isodose charts for electron beam 15 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

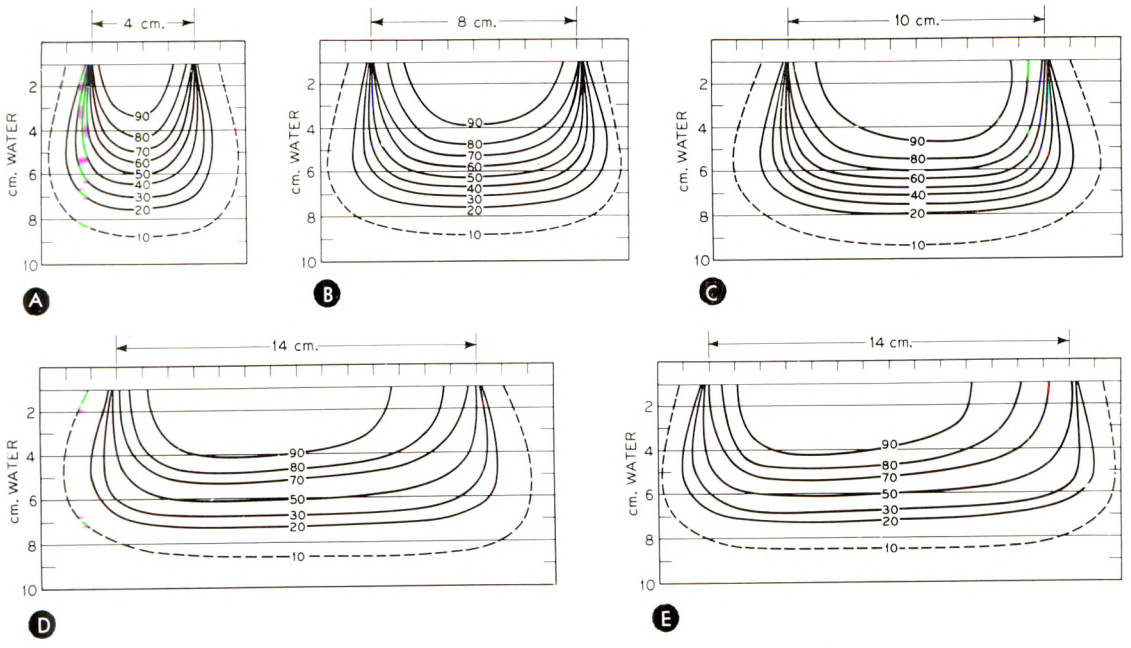


Fig. 5. Isodose charts for electron beam 18 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*L*) 14×7 cm.; and (*E*) 14×14 cm.

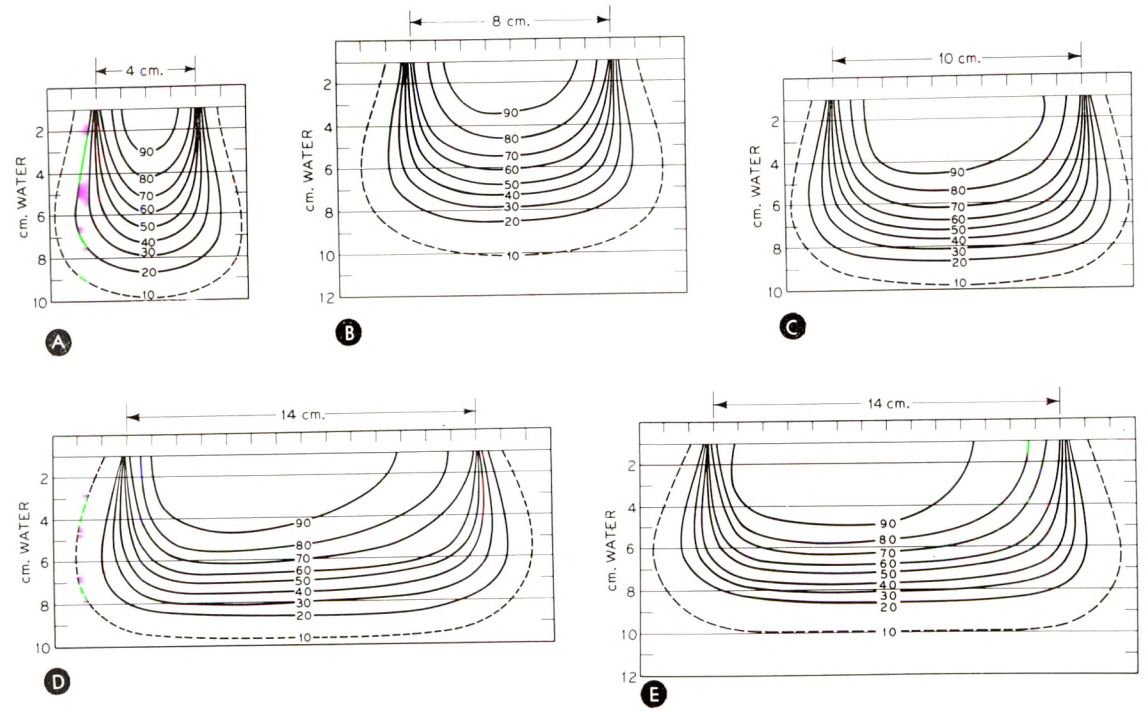


Fig. 6. Isodose charts for electron beam 21 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

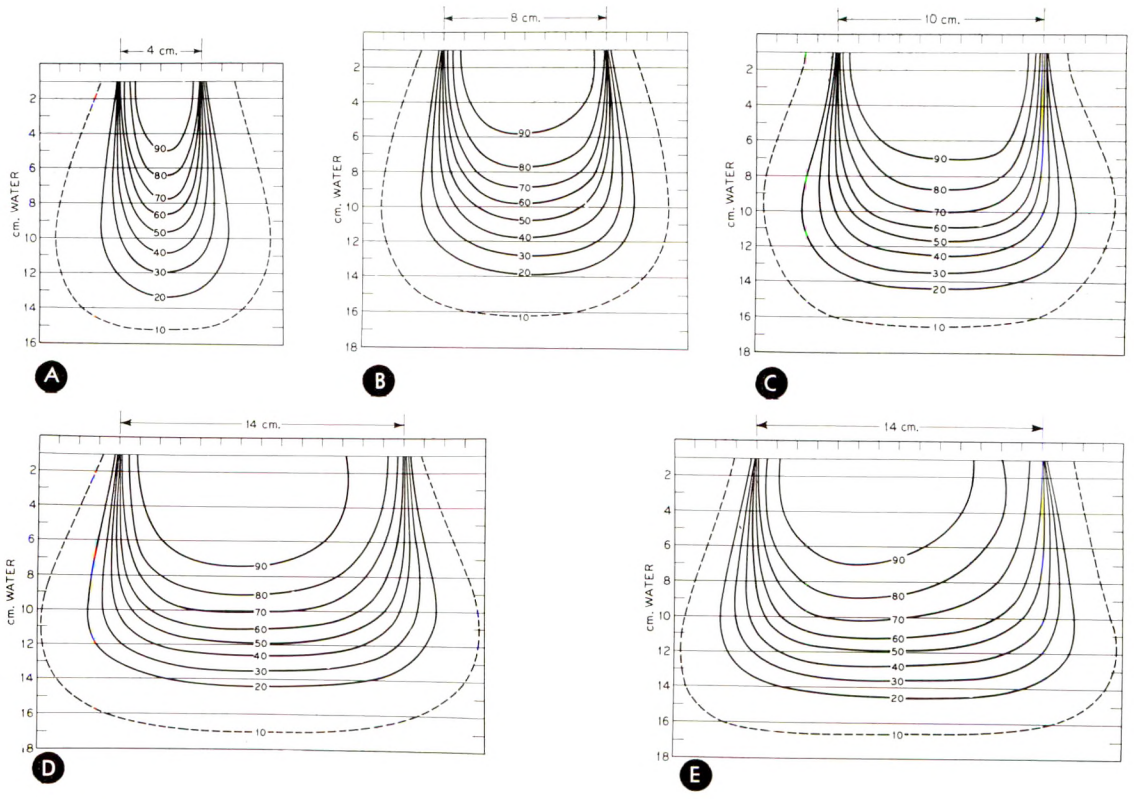


FIG. 7. Isodose charts for electron beam 32 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

Baldwin Dosemeter was compared and calibrated with our 25 r Victoreen chamber which was standardized by the National Bureau of Standards for the spectral energy of cobalt 60 radiation. Several measurements were taken for each particular depth in lucite and the results were reproducible within ±1.5 per cent. In addition, ionization measurements of the central depth doses were compared with photographic data for varying energies and field sizes, and good agreement between the methods was obtained (Fig. 1). Central depth doses were measured for all available cones at the following energies: 35 mev., 30 mev., 24 mev., 18 mev. and 10 mev.

TABLE V

ELECTRON BEAM CENTRAL AXIS DEPTH DOSE DATA

35 mev. Electron Beam; Scatterer 0.3 mm. Cu

Percentile	Depth of Percentile in cm. Water for Various Fields				
	4×4 cm.	4×8 cm.	8×10 cm.	7×14 cm.	14×14 cm.
90	5.6	6.0	7.5	7.4	7.5
80	7.0	8.0	9.7	9.3	9.9
70	8.2	9.3	11.2	10.6	11.5
60	9.4	10.5	12.3	11.8	12.7
50	10.7	11.7	13.3	12.9	13.5
40	11.9	12.8	14.2	13.9	14.2
30	13.1	13.9	15.2	14.8	15.0
20	14.4	15.2	16.0	15.7	15.8
10	16.3	16.5	17.0	16.7	16.9

Film was used to measure the isodose distribution of the electron beam in lucite. The advantage of film is the high spatial resolution. This is achieved by use of thin film and a densitometer which allows measurements of a spot of 1 mm. in diameter. A Photovolt Electronic Transmission Densitometer was used; the densitometric scale permitted the accurate reading to 0.01. Correlation between the density of the film

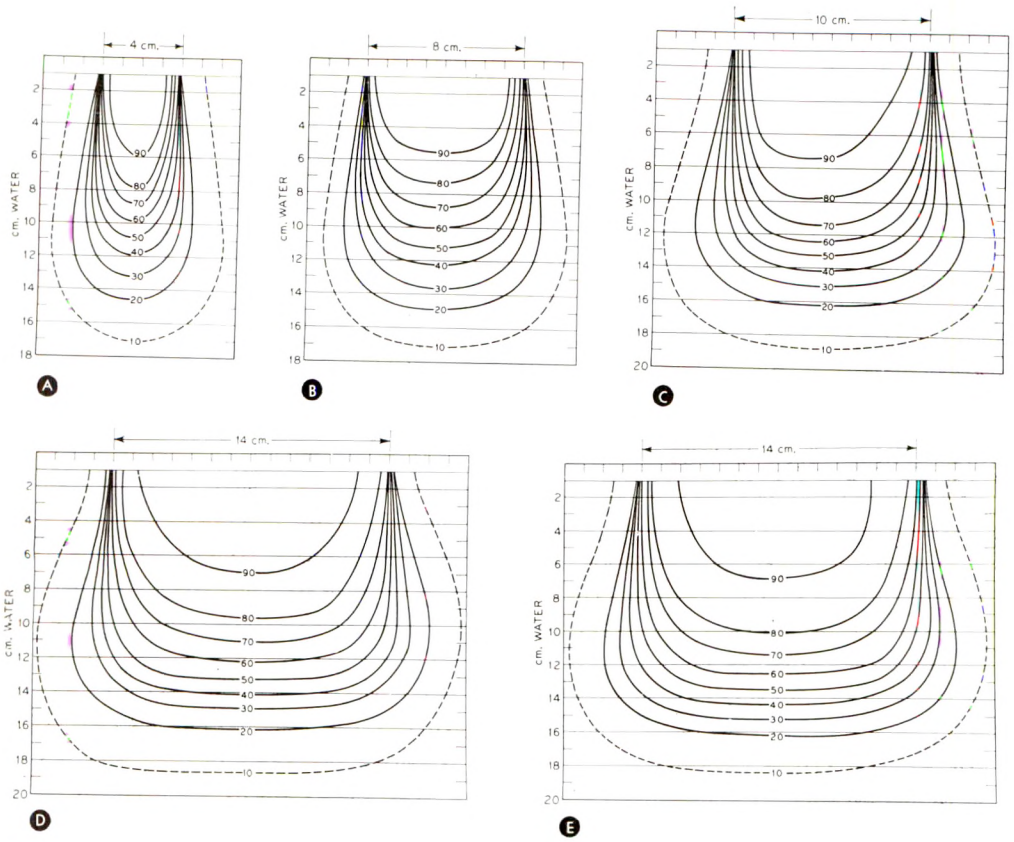


Fig. 8. Isodose charts for electron beam 35 mev., field sizes (*A*) 4×4 cm.; (*B*) 8×4 cm.; (*C*) 10×8 cm.; (*D*) 14×7 cm.; and (*E*) 14×14 cm.

and the dose may yield errors in excess of 10 per cent; however, it was found that, in relative dosimetry between different points of a film or of several films developed simultaneously, the error can be reduced to less than 5 per cent. DuPont Adlux film was used because of its low speed. The linearity of the density-exposure curve was tested for all energies and was found to hold up to a density of about 0.8–0.9. The low sensitivity of the film emulsion permits delivery of about 100 r for the maximum density on the linear part of the curve. The film, enclosed in a black envelope (to avoid Cerenkov radiation emanating from lucite at these energy levels), was placed parallel to the electron beam within the lucite phantom.[1] External pressure was applied to the phantom to prevent air gaps. Each group of films, exposed to a particular electron beam energy, was taken whenever possible from one large sheet of film to avoid variations in speed observed among different films from the same box. Each group was developed simultaneously under periodic agitation. Care was taken to hang the films in the developer in identical positions as far as the more exposed areas of the films were concerned. (Measurable differences have been observed between two identically exposed films when they were placed in the developer in different positions.)

DISCUSSION

Our initial purpose was to present isodose charts for the same group of electron beam energies as the central depth doses. However, it was found during our investigation that the films obtained at electron beam

energies of 24 mev. and 30 mev. showed a clearly visible band of density superimposed on the electron beam path, and extending far beyond the maximum range of the electrons. Our measurements indicate that this is caused by roentgen-ray contamination; it is presumed that some of the electrons hit the target at the moment of extraction from the doughnut. Strikingly, the same phenomenon, while observed consistently at energies from 22 mev. to 31 mev., is absent at all other energies. The film findings were substantiated by the shape of the central depth dose curves at 24 mev. and 30 mev., where increased levels of bremsstrahlung at the end of the electron ranges are evident. The level of the contaminating roentgen-ray component is calculated to be 10 to 12 per cent of the maximum electron beam output, assuming identical response of the film to the electron and roentgen-ray energies in the measured ranges. There is no final explanation of these phenomena, especially as they occur only in the middle ranges of the electron beam. Isodose determinations at 24 mev. and 30 mev. showed distortions and irregularities mainly in the middle and lower percentage levels. Treatment will not be carried out with these energies until the contamination is eliminated. Therefore, isodose charts for 21 mev. and 32 mev. have been substituted for the 24 and 30 mev.

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SCINTILLATION SCANNING OF THE LIVER*

I. PHYSICAL BASIS

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THE process by which the distribution of radioactive isotopes at points within the body is graphically or photographically recorded and which is known as scanning (or as scintillation scanning since all of this work has been carried out since the advent of scintillation counters) was first reported in 1951 by Cassen and associates.[8] Prior to the availability of the scanner as we know it today, scanning procedures or surveys were carried out by manually moving a radiation detector over selected areas of the body and recording the counts which were obtained at numerous positions.[15,16,17] This was a very time-consuming operation and did not afford as complete a description of the radioactive deposition as does the present mechanical scanner which automatically moves a detector over a given preselected area and records the data from an infinite number of sites. In addition to the increased data which are obtained using the mechanical scintillation scanner, this method also provides an unbiased and objective record of the deposited radioactivity.

The first organ which was subjected to the scintillation scanning procedure was the thyroid gland[1,2] since it offered an excellent opportunity of using the device and since there was a great interest in radioactive studies of this organ. These studies have continued since that time.

Following the successful use of the scintillation scanner in thyroid scanning, attempts were made to extend its usefulness to the scanning of other areas. Other organs which have been scanned successfully by this procedure are the gallbladder,[18] the liver,[4,5,9,10] the brain,[3,7] selected parts of the vascular system,[6,16] the spleen[11] and the kidneys.[13] Because of the much lower selective deposition of radioactive materials within these organs as compared to that found in the thyroid gland, the scanning procedures for these organs required refinements which had not been necessary for early thyroid scanning. The problems encountered in the scanning of certain of these organs, and the refinements which must be carried out to solve them, are described later in this paper. It is of interest at this point to examine the various types of information which scintillation scanning can provide. Included are data concerning size, shape, position, uniformity of isotope deposition and relationship of adjacent structures. It should be kept in mind that these data are obtained from differences in levels of radioactivity in the organ and surrounding areas, and that this radioactivity is truly what we are visualizing, and not the organ itself. In most of the organs listed above in which scintillation scanning has proved successful, the procedure has offered the only, or the best, visualization of the structures that was, or is, available. This has been especially true in the case of the liver for which no safe radiopaque contrast material or other means of delineation has been developed.

SCINTILLATION SCANNING REQUIREMENTS

The most important factor in all scintillation scanning procedures (and as a matter of fact, in all procedures in which localization of radioactive material is studied) is the

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† From the Department of Radiology of the University of Texas Southwestern Medical School and Parkland Memorial Hospital, Dallas, Texas.

so-called differential absorption ratio (DAR). This is the ratio of the amount of radioactivity absorbed or deposited in the area of interest to the amount of radioactivity deposited in immediately adjacent areas. It is obvious that as this ratio decreases, obtaining information about the deposition of radioactivity in the area of interest becomes increasingly difficult. This difficulty is seldom encountered in thyroid scanning due to the extremely high differential absorption ratio in the thyroid in most individuals. The range of differential absorption ratios obtained in the liver using I^{131} labeled rose bengal dye is generally from two to four, whereas the differential absorption ratio for the thyroid is usually in the vicinity of 100 or more.

Related to the differential absorption ratio is the actual level of radioactivity which can be obtained in the area of interest without exposing the patient to an excessive radiation dose. The lower the differential absorption ratio, the greater must be the administered dose of radioactive material in order to achieve a given level of deposition.

Assuming that a labeled compound having the optimal differential absorption ratio has been chosen and that the maximum acceptable dose has been administered to the patient, thus yielding the maximum obtainable deposition within the area of interest, the next problem is that of accepting as much information as is possible from this deposition of radioactive material. This must be accomplished in as short a time as possible so that the patient is not subjected to undue periods of immobilization. Although the maximum amount of information is striven for, this information must be of proper quality and must include data relating to only that portion of the scanned area from which it is obtained and for which it is subsequently recorded. This requires that we use a shielded detector and collimator which (1) resolve adjacent radioactive differentials with required precision, (2) have a high counting efficiency, and (3) properly shield out information from areas other than that "seen" by the detectors.

From the detector, information which has been accepted from the deposited radioactive material is transmitted to a data processing system. In this data processing system, the count information is modified in a number of ways before being passed on to the last part of the scanning system, the recorder. These modifications may include the following: (1) scaling-down of the count information; (2) pulse height discrimination; (3) count rate discrimination; and (4) contrasting. The scaling-down of count information (use of a scale factor of 8, 16, etc. counts per impulse to recorder) is used to "smooth" statistical variations and also to meet the speed requirements of the recording system. In both pulse height discrimination and count rate discrimination, unwanted or less useful information is discarded. (In each of these procedures, good or useful information may be accidentally discarded due to improper setting of the associated devices). Contrasting is accomplished by emphasizing differences in counting rate usually by means of an electronic circuit, and usually in a nonlinear manner.

Following the data processing system is the recording system which converts the information mechanically (solenoid tapper or "pen" and carbon paper), electrically (spark and teledeltos paper), or photographically (flashing light source moving over a film) into a "picture" or graphic representation of what the counter has "seen." It is at this stage of the scanning process that the need for data processing originates, since the eye is frequently unable to distinguish differences which actually exist in the information when it is recorded unprocessed. Many types of scanning information are completely unintelligible without modification and thus require data processing to be of value. It should be noted that the photographic type of recorder, because of the nature of the film response to light (Fig. 1), includes built-in count rate discrimination and contrasting. These modifications of data occur with film

because of the fact that film possesses a threshold below which it is unaffected by light and also because film density is not always a linear function of light intensity. If the levels of intensity of the light flasher and the variations in its intensity with counting rate can be adjusted, then these film properties can be used to advantage. Improper adjustment of the intensity and variations thereof can also work to the detriment of good scan visualization.

Most of the present data processing systems such as the count rate discriminators (background eliminator) and contrasters are able to emphasize differences in *two levels* of counting rate or radioactive concentration, but in doing so they often lose or distort beyond recognition other important or critical levels. It is because of this that the authors (in cooperation with Mr. William Romans of our Medical Electronics Section) have devised and are testing an experimental tape recording system which will allow display of the recorded information with a 60 to 1 speed-up. Scans which require an hour to record can be observed in one minute, and can be observed repeatedly in periods of one minute with different count rate discrimination levels. This will result in no loss or distortion of any information and without inconvenience to the patient or physician. It is felt that this system will allow the ultimate in visualization of all types of scintillation scanning information. Unfortunately, this new device was not available at the time when the data were obtained for Part II of this paper.

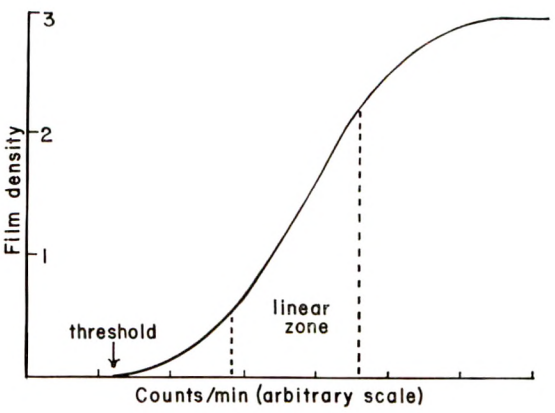


Fig. 1. Density versus counting rate for film exposed to a light source activated by radiation detection apparatus. Note the threshold, nonlinear portions, and linear portion of the curve. The steeper the slope of this curve, the greater the change in density per change in counting rate.

Scanning Equipment Used in These Studies

Early studies were carried out using a relatively simple but successful scanning apparatus. This consisted of a commercially available scanning frame with variable speed of travel and variable scan-line spacing, a $\frac{3}{4}$ inch by $\frac{3}{4}$ inch sodium iodide scintillation crystal, a special lead shielded straight bore collimator which provided a 12 mm. orifice and a minimum of 2 inch lead shielding in the forward and lateral directions. The signal from the scintillation counter was led directly into a simple binary scaler without pulse height selection (a $\frac{1}{16}$ inch thick lead shield was placed in front of the crystal to minimize entry of scattered I^{131} radiation). The scaler was used to scale-down the counting rate (scale of 8, 16, or 32 was generally used for dose of 4 μc/kg. body weight of I^{131} labeled rose bengal) prior to transmitting a signal to the recorder. The recorder consisted of two identical solenoid pens (tappers) used in conjunction with large size (14×17 inch) carbon paper and overlay paper. The signal from the scaler to one solenoid was direct, and thus recorded all impulses, whereas the pathway to the other solenoid passed through a count rate discriminator or background eliminator modified from the type described by MacIntyre and Houser.[12] This background eliminator was constructed so that it could be preset at a value of counting rate (determined by palpation and preliminary hand scanning by physician) below which impulses from the scaler were not allowed to reach the second solenoid. This eliminates from the second solenoid record all information below a certain carefully preselected counting

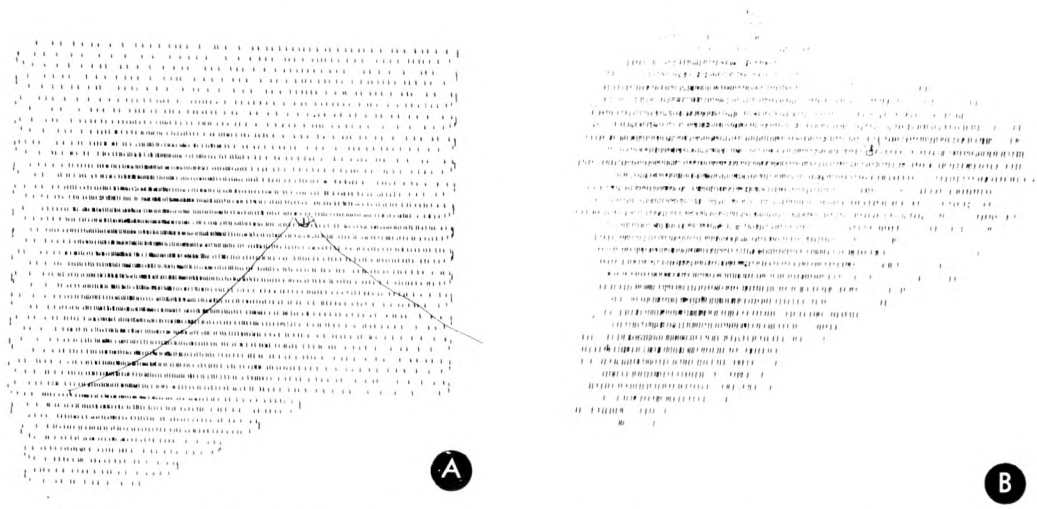


Fig. 2. Scintillation "dot" scan records made by two-solenoid scanner showing value of counting rate discrimination or background elimination. (*A*) Noneliminated scan of liver. (*B*) Eliminated scan of same liver showing defect in liver later proved to be carcinoma.

rate thus emphasizing certain differences in radioactive deposition. Figure 2, *A* and *B* shows noneliminated and eliminated scans. Information above the preselected counting rate is recorded by the second pen without distortion. The background eliminator control circuit was used with a time constant of approximately one quarter of a second so as not to have significant "memory" of counting rates from previously traversed scan areas. The double solenoid system is used so that in case of improper or inopportune settings of the background eliminator all available information is still available on the direct solenoid record. With this apparatus liver scans were run at the maximum speed of traverse (14 in./min.) and scan-line spacing was maintained at about $\frac{1}{4}$ inch—a usual liver scan required from one-half to one hour to complete.

Later (1961 to present) studies have been carried out using a greatly improved commercially available scanner which was further improved by our Medical Electronics Section (Fig. 3). This scanner uses a 3×1 inch sodium iodide crystal, a nineteen-hole focusing collimator with minimum 2 inch forward shielding, a pulse height selector (ordinarily used with integral selection at about 150 kev.), a photographic recording system, and a background eliminated two-solenoid tapping recorder. The photographic recorder consists of a light source (1 inch cathode ray tube with fluorescent face which moves over a piece of usual roentgenographic film). The light intensity emitted by this tube can be adjusted to any level for any counting rate and its brightness can be caused to increase at different rates for different changes in counting rate. The first adjustment can be used to eliminate information below a certain level, and the second adjustment can be used to emphasize differences in counting

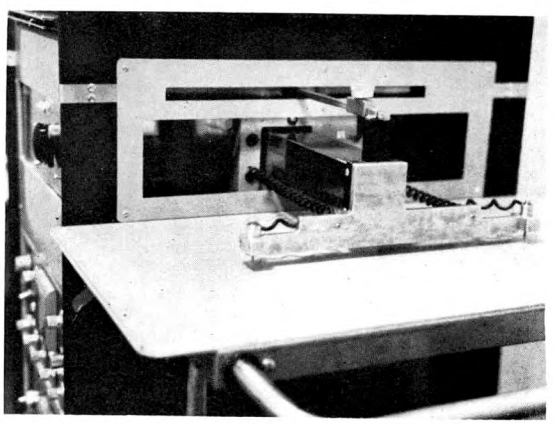


Fig. 3. Photograph of presently used scintillation scanner showing two-solenoid modification.

rate by effectively changing the slope of the density versus the counting rate curve (Fig. 4).

Examination of curves in Figure 4 will show that for steep slopes (high contrast), only a limited range of counting rates can be distinguished, whereas gentle slopes allow differentiation of a wide range of counting rates, but with less contrast. For steep slopes, statistical variations in counting rate are emphasized and must be allowed for in examining photographic scan records. From the foregoing, it is evident that high contrast settings can provide discrimination between small differences in counting rate, but that because of inherent difficulties, this type of setting should usually be avoided. The two-pen mechanical recording system was added in our institution in place of a single electrical recorder. The background eliminator circuit in the instrument was also modified to have a shorter time constant and thus a decreased "memory."

With this latter scanner, it has been our experience that the graphic and photographic scan records tend to supplement each other and that *both* should be included and viewed as of equal importance. The "dot" scan data also are of great value when one wishes to superimpose the scan on a roentgenographic image in order to compare the two records. It is our opinion that the tape recording system described briefly above, when used in conjunction with a resettable linear type of data processer, will allow for the most accurate discriminat on of multiple counting rate levels. The record from this and other systems is probably best displayed and interpreted on photographic film when superimposition on roentgenographic film is not required.

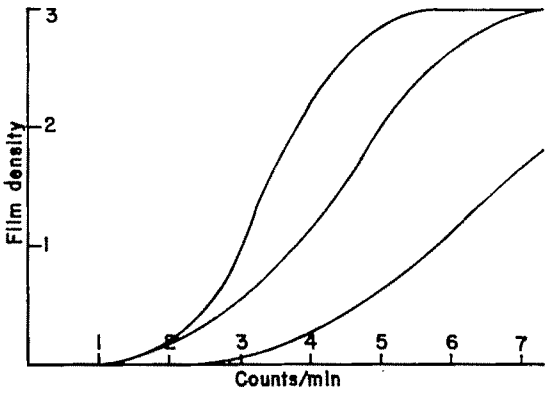


Fig. 4. Series of typical density versus counting rate curves for photographic recording system. Curves show various threshold positions and various density versus counting rate slopes. Note the narrow range of counting rates measurable on the linear portion of the steep curves as compared to the wide range of counting rates measurable on the less steep curves.

SUMMARY

1. Scintillation scanning of any organ requires the use of a radioactively labeled compound having a relatively high differential absorption ratio (DAR) in the organ of interest. Preferably this differential absorption ratio should be two or more.

2. Proper scintillation scanning procedures for any organ require the accumulation of the maximum amount of data available from the site of radioactive deposition, but require that this information be obtained only from the area in question and not from adjacent areas. This necessitates the use of a well collimated, highly efficient, well shielded scintillation counter. The authors are at present using a 3×1 inch sodium iodide crystal, a 19 hole focusing collimator, and a 2 inch minimum lead shield in the forward direction.

3. Data from the radiation detector must pass through a data processing system prior to being transmitted to the recording system. This data processing system is used to scale-down count information in order that it be compatible with the speed of the recorder; to subject the incoming signals to pulse height analysis in order to eliminate low energy scattered radiation; and to either modify the incoming signals by means of a count rate discrimination or to apply a contrasting system in order to emphasize differences in deposition of radioactive materials. The authors are presently using a scintillation scanning system which provides both a photographic recorder and a dual solenoid "dot" scan recorder. Integral

pulse height discriminated signals are fed into both recording systems. Single or unscaled-down pulses are fed into the photographic system, while scaled-down (scale of 8, 16, 32, etc.) pulses are fed into the "dot" recording system via a counting rate discrimination or background elimination circuit. Both recording systems provide for emphasizing small count rate differences by different mechanisms. It is the opinion of the authors that both graphic and photographic recordings be viewed with equal diligence since they have been found to supplement each other.

4. An experimental tape-recorder scintillation scanner is presently being tested by the authors. This system allows play-back of the recorded data at a speed-up factor of 60 to 1. This rapid play-back allows one to observe the scanning data from a scan which takes one hour to record in a period of one minute. The data can be replayed as many times as required and at as many different data processing settings as are desired. The present data processing system on this experimental scanner is a modification of the counting rate discrimination circuit. The authors feel that this system with its capability of multiple replay at various count rate discrimination settings offers the best opportunity for examining many small differences in radioactive deposition in a given scan, since it provides for a completely linear reproduction of the data which have been obtained.

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SCINTILLATION SCANNING OF THE LIVER*

II. CLINICAL APPLICATIONS

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EVEN in the young field of nuclear medicine scintillation scanning of the liver is a relatively new technique. The basic principles were first described in 1953[17–19] and yet, although the test is intensively performed in a few centers,[1–16,21] it is still not widely practiced. In institutions where it is available, however, it rapidly becomes a valuable part of the clinical diagnostic program. In a busy 600 bed general hospital, such as our own, scintillation scanning of the liver may be performed 200 to 250 times a year for diagnostic purposes. In this paper we will attempt to outline some of the principal areas of usefulness of this interesting test.

In the preceding paper[11] we have reviewed the theory and instrumentation of liver scanning, and have considered some of the experimental aspects of scan data processing. The scans included in this paper and derived from our routine clinical program were made with one of two commercial instruments, both of which were modified in our laboratory. Our first scanner employed a 1×1 inch thallium-activated sodium iodide crystal probe and a special lead collimator which contained a 12 mm. cylindrical opening. This collimator afforded no less than 2 inches of lead shielding over the sides and front surface of the crystal. A background eliminator[13] was an essential part of this unit. Scan data were presented in pictorial form by a two-pen solenoid printer system which made records on translucent tracing material by striking through carbon paper. One solenoid recorded all counts detected by the scanning probe, while the other presented only information which had been processed by the background eliminator.

Our second commercial scanning unit, acquired within the past year, has for its detector a 3×1 inch thallium-activated sodium iodide crystal with appropriate shielding and a focusing collimator. This unit has two alternative recording systems. One of them is the background eliminator and dual-solenoid circuit described above, while the other employs a contraster circuit and exposes roentgenographic film by means of a cathode ray tube and fluorescent screen. Both mechanical prints and photoscans will be presented throughout this paper. In general, we have observed that photoscans are more readily interpreted by those trained in deciphering the densities of roentgenograms, but mechanical prints can be interpreted as readily with practice.

For each photoscan shown below we have also made a background-eliminator-processed scan. It has proved to be of value to have both of these data treatment systems available, for more information may be gained by examining both records together than by examining either separately.

Tracers employed for scintillation scanning of the liver include substances which are selectively removed from the blood stream by the hepatic parenchymal cells or, alternatively, by the reticuloendothelial, or Kupffer's cells. I^{131}-rose bengal[20] and I^{131}-

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cholografin* are the principal tracers used for visualizing liver parenchymal cells and each may be used in amounts of from 3 to 5 μc/kg. of the patient's body weight. Radioactive colloidal gold (Au^{198}), which is deposited in the cells of the reticuloendothelium, is preferred as a tracer by several groups[8] which use it in amounts of 100 to 400 μc. Neither type of tracer is wholly ideal. The drugs which accumulate in the hepatic parenchymal cells each have a relatively short biologic half-life. As a result, the concentration of tracer within the organ of interest actually undergoes a change during the interval of the scan, approximately thirty to forty-five minutes, but we do not feel that this concentration difference militates against the use of this group of agents. Radioactive colloidal gold, while it does not undergo material change in concentration after equilibrium is reached, is not as effective in portraying disease states which affect the parenchymal cells, such as hepatitis and cirrhosis. With tracer material of either sort, the whole body and liver radiation doses are of the order of those which might be delivered in a radiologic examination which includes image-intensification fluoroscopy.

Information can be derived from liver scanning about the size, shape, position and internal configuration of the liver.[5] Scanning will continue to provide the best information of this sort until the development of a safe radiopaque contrast material which is specifically deposited in the liver. No such material is in immediate prospect, however. The alternative methods, intrahepatic or intrasplenic portal venography, afford similar information, but they are not always diagnostic, and they are not without hazard to the patient.

THE NORMAL LIVER

Scintillation scanning of the liver is a radiographic technique, and, as in the case of all other such techniques, interpretation of the abnormal is based upon a sound knowledge of normal variations.

* Courtesy of Dr. Paul Numeroff, E. R. Squibb & Sons.

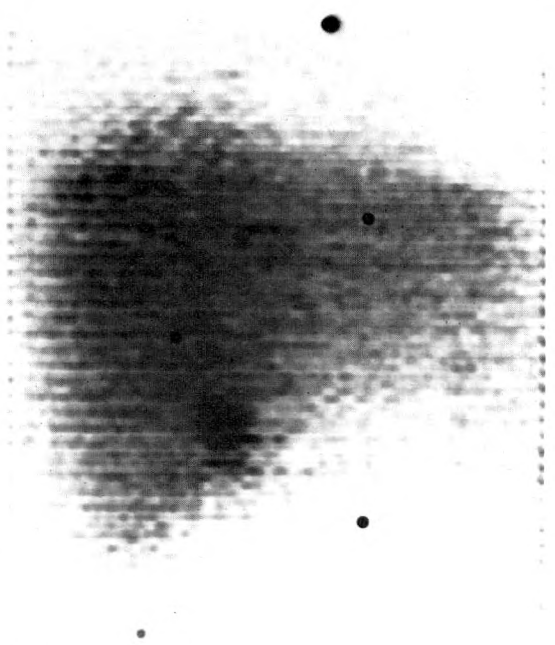


Fig. 1. Photoscan of a normal liver. Slight variation in internal pattern is quite within normal limits. Note gallbladder.

Figure 1 shows the photoscan of a normal liver of classic configuration. The slight variation in photographic density within the liver is normal.

Figure 2 is a scan made by the solenoid printing system in which each printed stroke represents a certain number of counts of activity, usually not less than 16. Thus, the closer together the strokes are spaced the more activity is present in a given area. The normal liver seen in Figure 2 has a relatively short left lobe, not extending past the midline, but an elongated right lobe, which on a matching roentgenogram extended below the crest of the ilium.

Figure 3 shows a large caudate lobe extending inferiorly below the left lobe of the liver, well down into the left upper quadrant of the abdomen. This liver was examined in the course of a laparotomy, and was found to be entirely normal.

Changes in size, shape and position of the liver may occur over a short period of time in the course of recovery from acute liver disease, as is illustrated in Figures 9, *A* and *B;* and 12, *A* and *B*. The liver may in-



FIG. 2. Solenoid print scan of a normal liver of different configuration from that in Figure 1. Curved line and cross denote the xyphoid process. Note that the left lobe is short and does not extend much beyond the midline, while the right lobe is relatively long. On a matching six-foot, anteroposterior, midrespiration roentgenogram of the abdomen, the shadow of the right lobe was superimposed upon that of the crest of the ilium.

crease in size with the progression of space-occupying neoplastic disease (Fig. 8 and 14), and changes in the contour of an otherwise normal liver may occur if it is displaced or rotated (Fig. 4, *A* and *B;* and 7). As a rule, however, information on changes in size and shape is of only incidental value. A far more useful parameter is position.

The liver may be displaced inferiorly by space-occupying processes above the diaphragm. Figure 4 shows a liver scan (*B*) and a six-foot upright posterioranterior chest film roentgenogram (*A*). Physical examination of the patient revealed a large, smoothly rounded mass in the right upper quadrant, well below the costal margin. The scan shows a liver of approximately normal size, shape and internal structure lying wholly below the costal margin, indicated on the scan by the curved solid line. The liver has been displaced from its normal situation in the right upper quadrant as a consequence of inversion of the diaphragm by a tension hydrothorax.

The liver may be displaced from its customary position by abdominal disease of several different types. In the presence of ascites the liver may "float" away from the right lateral body wall and often may lie in the upper abdominal midline, as in Figure 5.

Localization of the true position of the liver assumes its maximal importance, however, in the differential diagnosis of apparent elevation of the right leaf of the diaphragm. Certain preliminary tests may be applied, such as lateral decubitus roentgenograms to rule out subpulmonary pooling of right pleural fluid, but if these do not yield definitive information, then the liver scan may prove to be of value.

The hepatoscan is made in the usual way and is then superimposed upon a six-foot, anteroposterior, recumbent, midrespiration roentgenogram of the thoracoabdominal region, matching certain geographic landmarks drawn upon the scan (Fig. 6) with radiopaque film markers placed at these same points. Figure 6 shows a liver displaced from the right leaf of the diaphragm by a space-occupying process later identified surgically as a large subdiaphragmatic

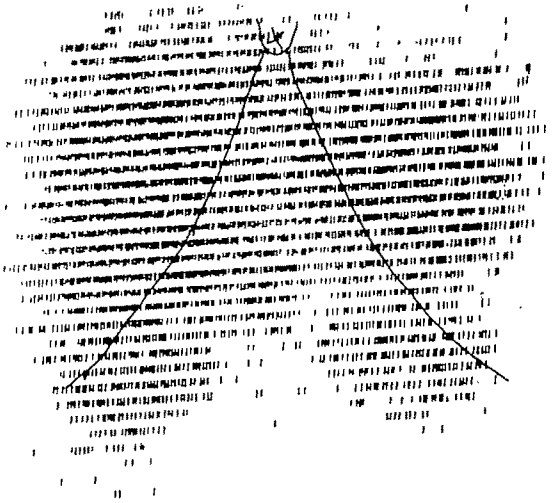


FIG. 3. Solenoid print scan of a normal liver from which arises a large caudate lobe. Neither this lobe nor the right lobe was palpable beneath the costal margin, demarcated by solid lines.

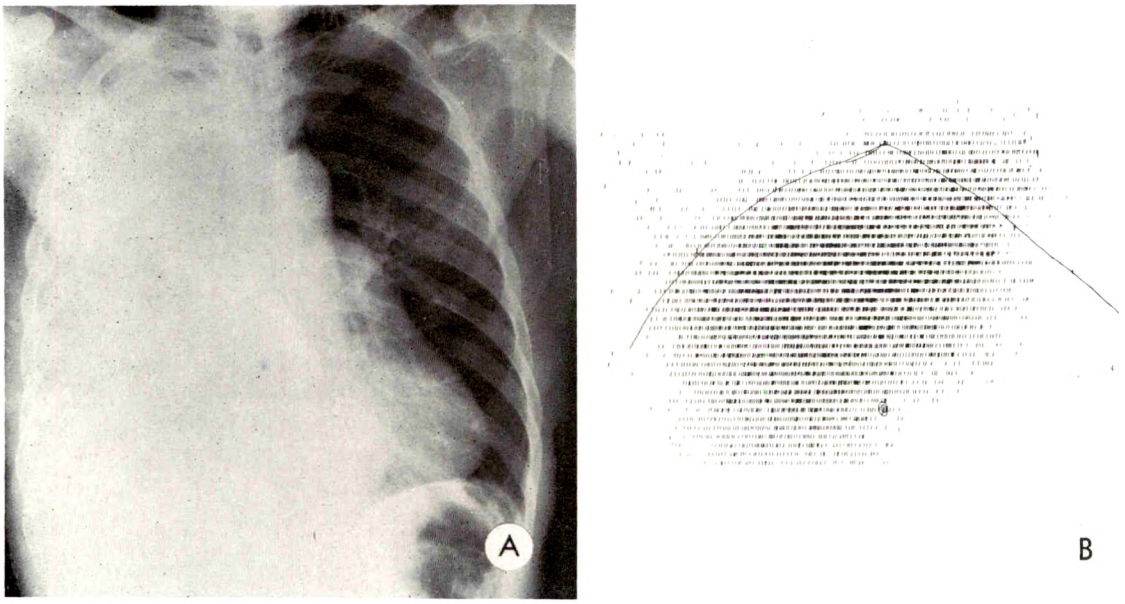


Fig. 4. (*A*) Six-foot upright posteroanterior chest roentgenogram shows the right hemithorax to be opaque and the mediastinal structures to be displaced slightly to the left in a patient known to have bronchogenic carcinoma with tension hydrothorax. We were unable to obtain the usual six-foot anteroposterior, recumbent matching roentgenogram. (*B*) Solenoid print shows a liver of relatively uniform pattern but of somewhat unusual shape, presumably due to rotation. It lies wholly below the costal margin (solid line) and was palpable as a smoothly rounded mass in the right upper quadrant of the abdomen.

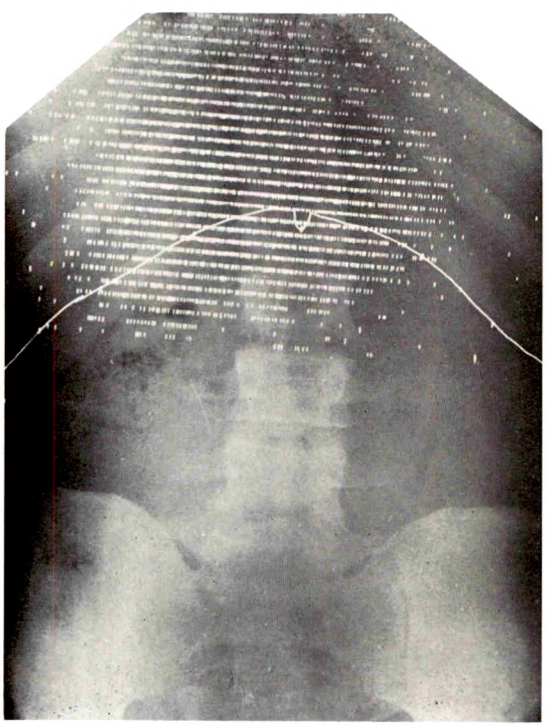


Fig. 5. Hepatoscan superimposed upon a matching six-foot, anteroposterior, recumbent, midrespiration roentgenogram of the abdomen shows the liver in the upper abdominal midline in a patient known to have massive ascites of neoplastic origin. Solid line represents costal margin. The liver often floats into this midline position in patients with voluminous ascites.

abscess occurring after spontaneous rupture of the gallbladder.

In Figure 7 the space under an elevated right diaphragmatic leaf is occupied medially by a small, rotated liver, and laterally by a large, multiocular subdiaphragmatic abscess.

Figure 8 shows the right leaf of the diaphragm to have been elevated by a greatly enlarged liver. The irregular pattern and many blank spaces within the liver were produced by a huge hepatoma.

In our laboratory, we prefer to use background-free mechanical prints on translucent paper for superimposition upon match-

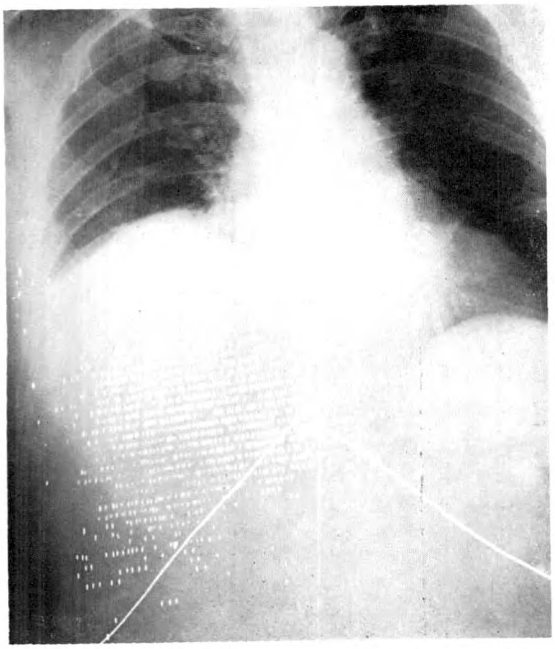


Fig. 6. Hepatoscan is superimposed upon a matching six-foot, anteroposterior, recumbent, midrespiration chest roentgenogram using markers on scan and film. The solid line represents the costal margin. The liver, of somewhat nonuniform pattern, is displaced from the shadow of the right leaf of the diaphragm by a process later identified surgically as a subphrenic abscess. (Reprinted by permission of the *Southern Medical Journal.*)

ing roentgenograms of the chest and abdomen when there is a question of displacement of the liver, for the reason that they obscure less roentgenographic detail than do photoscans.

CHANGES IN STRUCTURE OF THE LIVER

In our experience of approximately 1,200 liver scans, we have as yet found no space-occupying lesion within the liver capable of

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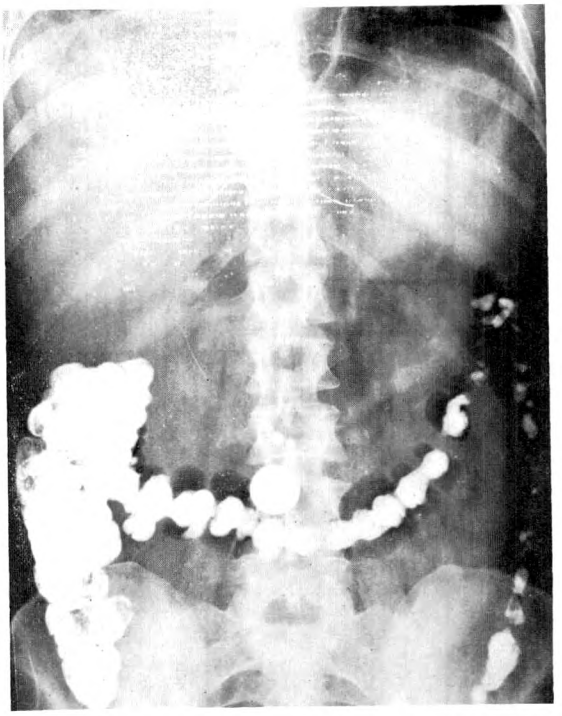


Fig. 7. Hepatoscan superimposed upon a six-foot, anteroposterior, recumbent, midrespiration roentgenogram shows the space under an elevated right diaphragm to be occupied medially by a liver which appears somewhat small, perhaps due to rotation. The space-occupying process seen laterally was identified surgically as a subphrenic abscess.

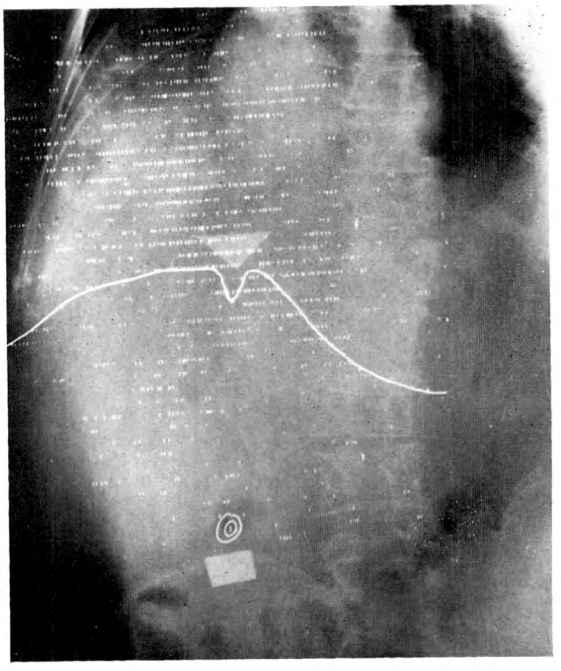


Fig. 8. Hepatoscan is superimposed upon a matching roentgenogram, made in the usual way, except in the right posterior oblique position, due to the fact that the patient was in pain and was unable to lie supine. The space under an elevated right diaphragm is occupied entirely by a large liver of markedly nonuniform pattern. Needle biopsy specimen yielded the diagnosis of hepatoma. Reorientation markers on the roentgenogram and scan are seen to the right of the spine.

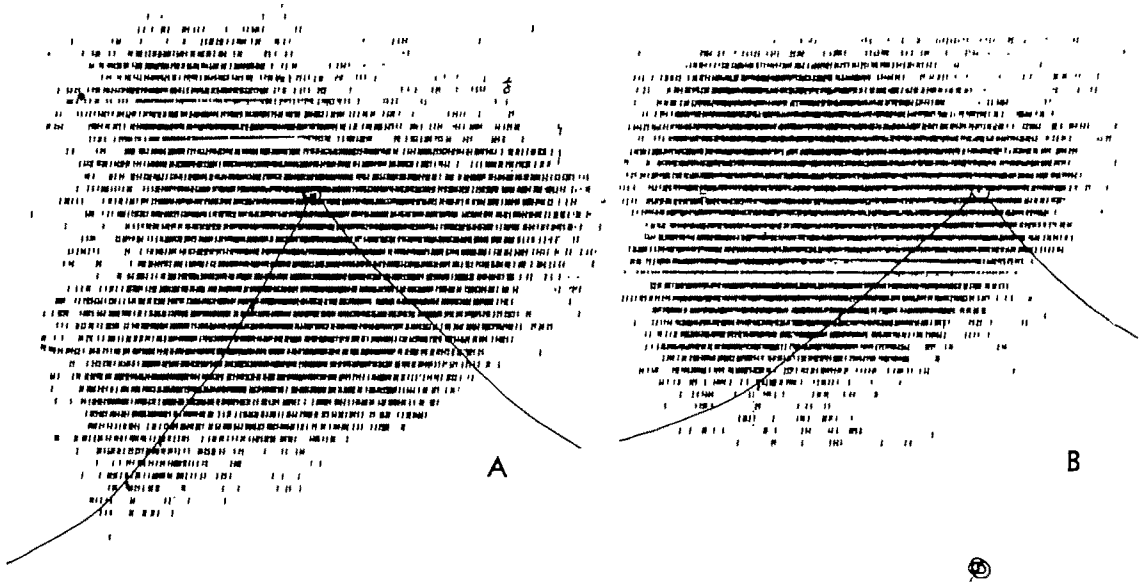


FIG. 9. (*A*) The liver is somewhat enlarged and extends well below the costal margin (solid line). A defect is seen in the upper portion of the lateral aspect of the right lobe, representing localized abscess formation in a patient with amebic hepatitis. After a course of antiamebic therapy, a follow-up scan (*B*) shows reduction in the size of the liver and disappearance of the abscess defect. (Reprinted by permission of the *Southern Medical Journal*.)

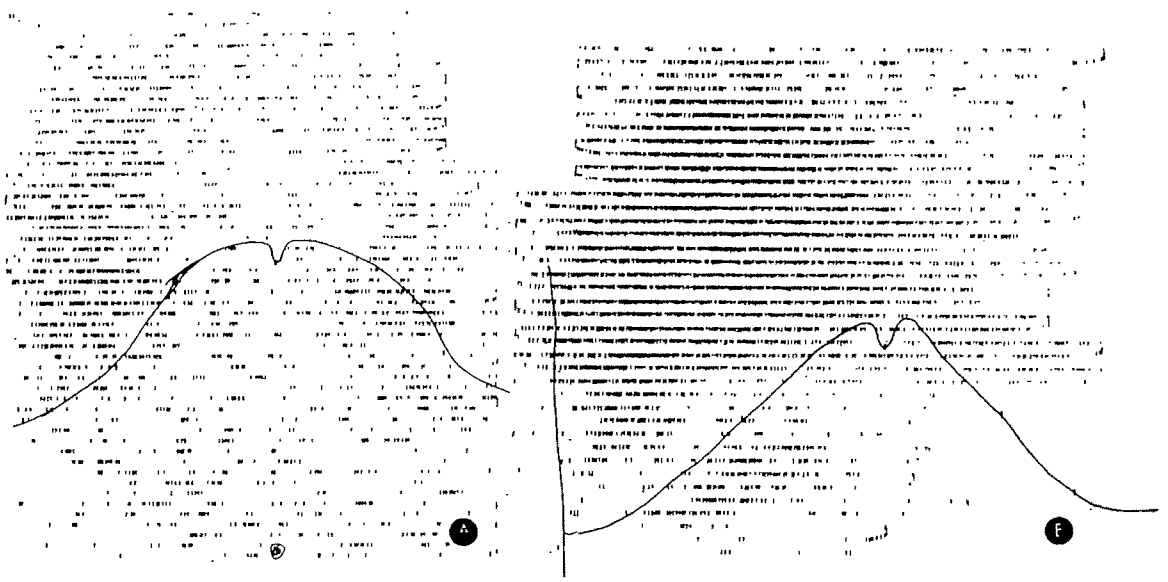


FIG. 10. (*A*) Hepatoscan shows a huge central defect with little normal pattern identifiable except along the lateral aspect of the right lobe. Curved line represents costal margins and the concentric circles the umbilicus. (*B*) Scan made three weeks after the surgical removal of 1,500 cc. of pus from an amebic abscess cavity shows a return of liver pattern to a more normal state and disappearance of the large defect. Solid line represents the costal margin and right lateral body wall.

taking up significant amounts of I^{131}-rose bengal, not even the most well-differentiated of hepatomas. Therefore, all mass lesions within the liver apparently will create a defect, or hole, within the normally uniform scan pattern. The edges of the liver, as seen on a scan, are usually somewhat irregular, especially along the lateral aspect of a thin left lobe, making these areas difficult to interpret, but areas of diminished activity which occupy two adjacent lines of the scan in the central portion of the liver may be presumed to be caused by non-liver tissue. The limitation of the scan method in detecting such lesions is still somewhat severe. While nodules approximately 1.5 cm. in diameter may be found in the dome of the liver or in its left lobe, those buried posteriorly within the substance of the thick right lobe may attain a size of 2.0 to 3.0 cm. and still escape detection. This limitation may be overcome to some extent by use of a curved-path scanner,[12] or by employing multiple settings of a background eliminator, or several background eliminators, as was brilliantly illustrated by MacIntyre and his associates,[14,16] but in the main it is felt that maximum information will not be extracted from the hepatoscan without some sort of data processing system such as that outlined in the preceding paper.[11]

In general, inflammatory and malignant neoplastic lesions are those most frequently responsible for hepatoscan defects. In rare instances developmental abnormalities, such as cysts or hemangiomas, may be encountered.

Defects in the liver are, of course, produced by surgical removal of portions of its substance after trauma. The scan is important in estimating hepatic regeneration and the viability of damaged segments, but this is a somewhat sizable field in itself, and will be described in detail in a later publication.

Inflammatory lesions may cause solitary defects, such as that seen along the lateral margin of the right lobe of a somewhat enlarged liver in the scan in Figure 9 *A*. The clinical course of this patient indicated the enlargement of the liver to be the result of amebic hepatitis and the right lobe defect to be a localized amebic abscess. In Figure 9 *B*, a post-treatment scan, disappearance of the defect and return of the liver to normal size is noted.



Fig. 11. Numerous small defects throughout the liver in a patient known to be in the fatty stage of Laennec's cirrhosis. Solid line represents the right costal margin and the concentric circles the xyphoid and umbilicus.

In Figure 10 *A* there is a huge central defect caused by an amebic liver abscess from which 1,500 cc. of pus was evacuated surgically. Figure 10 *B* shows return to a more normal pattern and size after drainage.

Acute liver disease, such as infectious hepatitis or the florid state of Laennec's cirrhosis, may produce not only a certain amount of enlargement of the liver, but also a pattern of diffuse, patchy defects (Fig. 11). It should be pointed out that diffuse metastases to the liver may also present such an appearance (Fig. 17).

Figure 12 *A* shows the diffusely mottled pattern of the liver of a patient known to have acute infectious hepatitis. Figure 12 *B* shows a scan made after recovery, some three months later, in which the liver is seen to be of smaller size, and of a normally compact, uniform pattern.

Both hepatomas and metastases from extrahepatic malignant neoplasms may create solitary defects. The photoscan in Figure 13 shows a large solitary metastasis

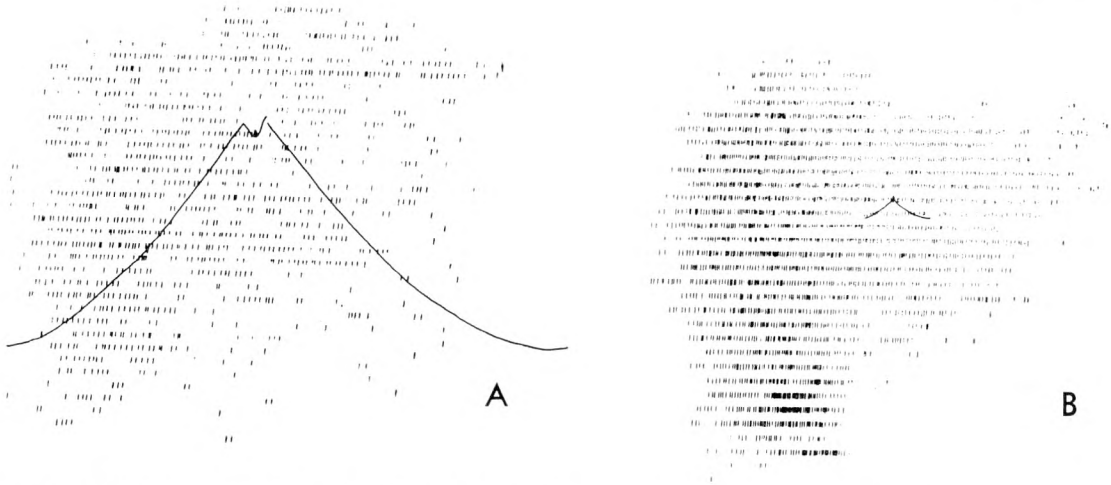


Fig. 12. (*A*) Numerous defects throughout the liver in a patient known to have acute infectious hepatitis. Solid line represents costal margin. (*B*) Scan made six weeks later shows return of a normal pattern after recovery from infectious hepatitis. Area of increased count at the tip of the right lobe medially represents the gallbladder.

occupying the dome of the liver on the right side. Needle biopsy showed undifferentiated carcinoma of unknown primary site.

Figure 14 shows a large metastasis from an embryonal carcinoma of the testis which replaced much of the right lobe of an enlarged liver.

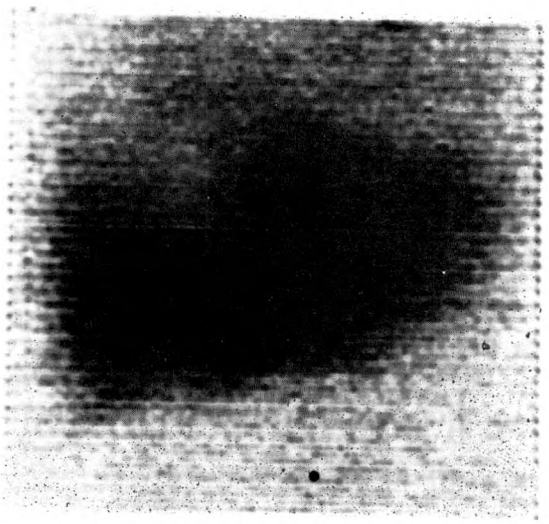


Fig. 13. Photoscan shows a round area of decreased density in the dome of the liver on the right side caused by a metastasis from an undifferentiated carcinoma of unknown origin, diagnosed by needle biopsy.

A hepatoma has created the large central defect which is seen in Figure 15.

Multiple large defects as seen in Figure 16 are usually the result of the presence of numerous tumor metastases. The primary tumor here was a bronchogenic carcinoma.

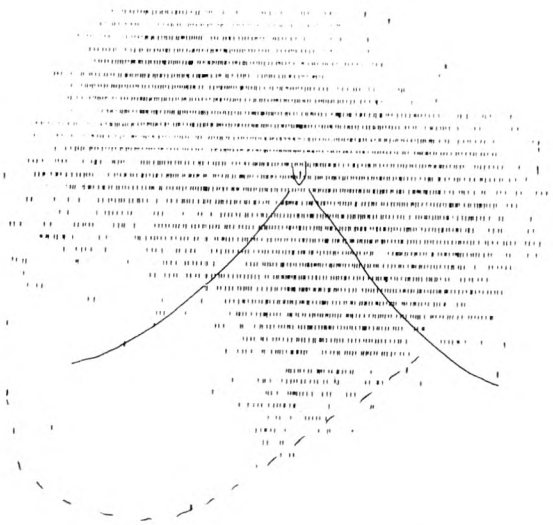


Fig. 14. The inferior portion of the right lobe of the liver is occupied by a large metastasis from a known embryonal carcinoma of the testis. Dotted line represents the margin of palpable liver, and the solid line the costal margin. (Reprinted by permission of the *Texas State Journal of Medicine*.)

Figure 17 shows a pattern of numerous small defects throughout the liver caused by multiple metastases from a carcinoma of the pancreas. Note the similarity between this pattern and that of acute hepatic inflammatory disease as seen in Figure 12, *A* and *B*.

The liver has an amazing capacity to sustain life, even when replaced almost entirely by neoplasm. Figure 18 shows three islands of functioning tissue remaining in a huge liver diffusely involved by metastases from an adenocarcinoma of the cecum. Even more remarkable is the fact that the patient whose scan is illustrated in Figure 19 continued to survive, even though his hepatoma had replaced virtually all of his hepatic parenchyma.

Liver scanning has been found useful in preoperative evaluation of the patient thought to have neoplastic disease. In a number of instances otherwise undetectable metastases have been demonstrated before laparotomy by the hepatoscan technique.[5,6] The test may also be employed to follow the course of a patient who has been treated for a neoplasm. For example, Figure 20 *A*

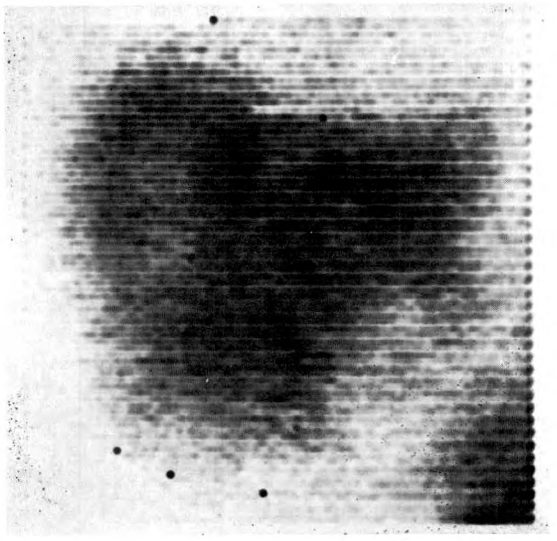


Fig. 16. Photoscan shows many areas of diminished density throughout a liver of markedly irregular shape caused by metastases from a bronchogenic carcinoma. Dots in the right lower quadrant represent the lower margin of a palpable mass. Upper midline dot represents the xyphoid process. In the left lower quadrant there is an accumulation of tracer in the bowel, a residual of an unsatisfactory hepatoscan performed on the previous day.

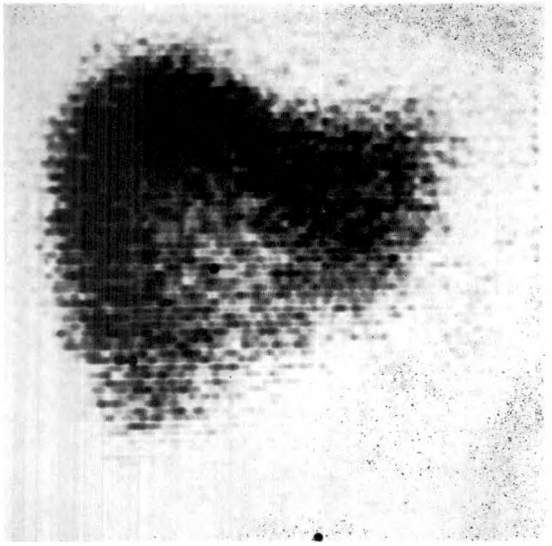


Fig. 15. Photoscan shows a large central space-occupying lesion, proven by needle biopsy to be a hepatoma. Black dots superimposed upon the liver represent the costal margin, and the lower central dot the umbilicus.

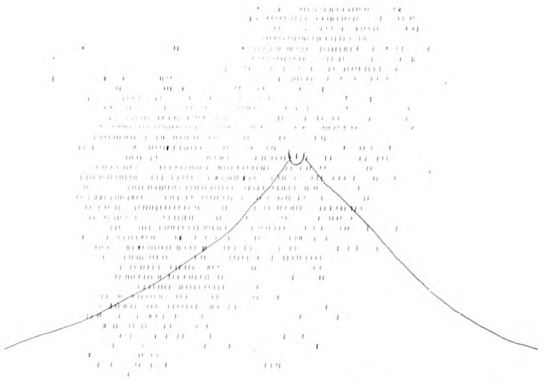


Fig. 17. Multiple, rather uniform defects throughout the liver of a patient known to have numerous metastases from a carcinoma of the body of the pancreas. Note the similarity between this pattern and that of florid cirrhosis (Fig. 11) and acute liver disease (Fig. 12, *A* and *B*). Solid line represents the costal margin. Pattern of strokes above and to the left of the liver represents cardiac blood pool. (Reprinted by permission of the *Southern Medical Journal.*)

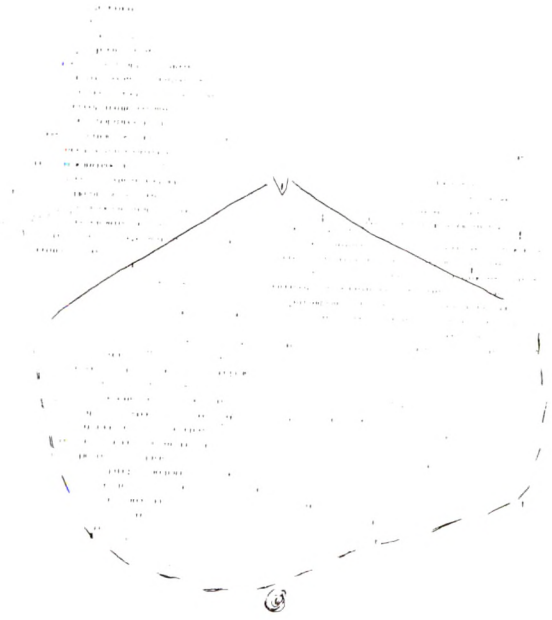


Fig. 18. Only a few islands of active liver tissue remain in a liver otherwise occupied by metastases from an adenocarcinoma of the cecum. Solid line represents the costal margin, the dotted line the inferior margin of a palpable mass, and concentric circles the umbilicus.

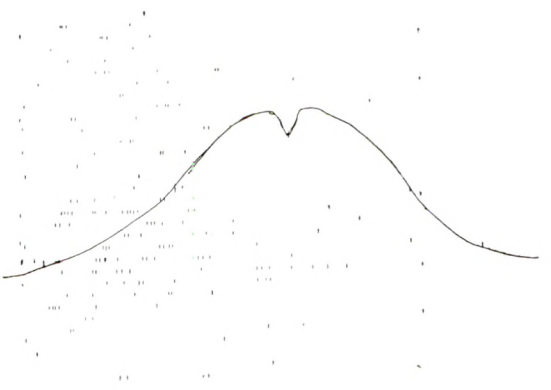


Fig. 19. Virtually no identifiable liver tissue remains in the presence of a large hepatoma. Solid line represents the costal margin.

is the scan of a patient who had a roentgenologically suspected colon carcinoma. This scan was done preoperatively to determine whether metastases were present, and the examination was reported as a negative one. On a scan performed five months after surgical removal of the bowel cancer (Fig. 20 *B*), there is ample evidence of replacement of much of the left lobe by metastatic



Fig. 20. (*A*) Preoperative hepatoscan of a patient with roentgenographically demonstrated carcinoma of the left colon. Despite the presence of a somewhat irregular pattern in the left side of the dome of the liver, this was interpreted as a negative examination. (*B*) Many defects are present in the dome and left lobe of the liver in a scan performed five months after surgical removal of the left colon carcinoma. At autopsy these were shown to represent metastatic disease. Solid line represents the costal margin.

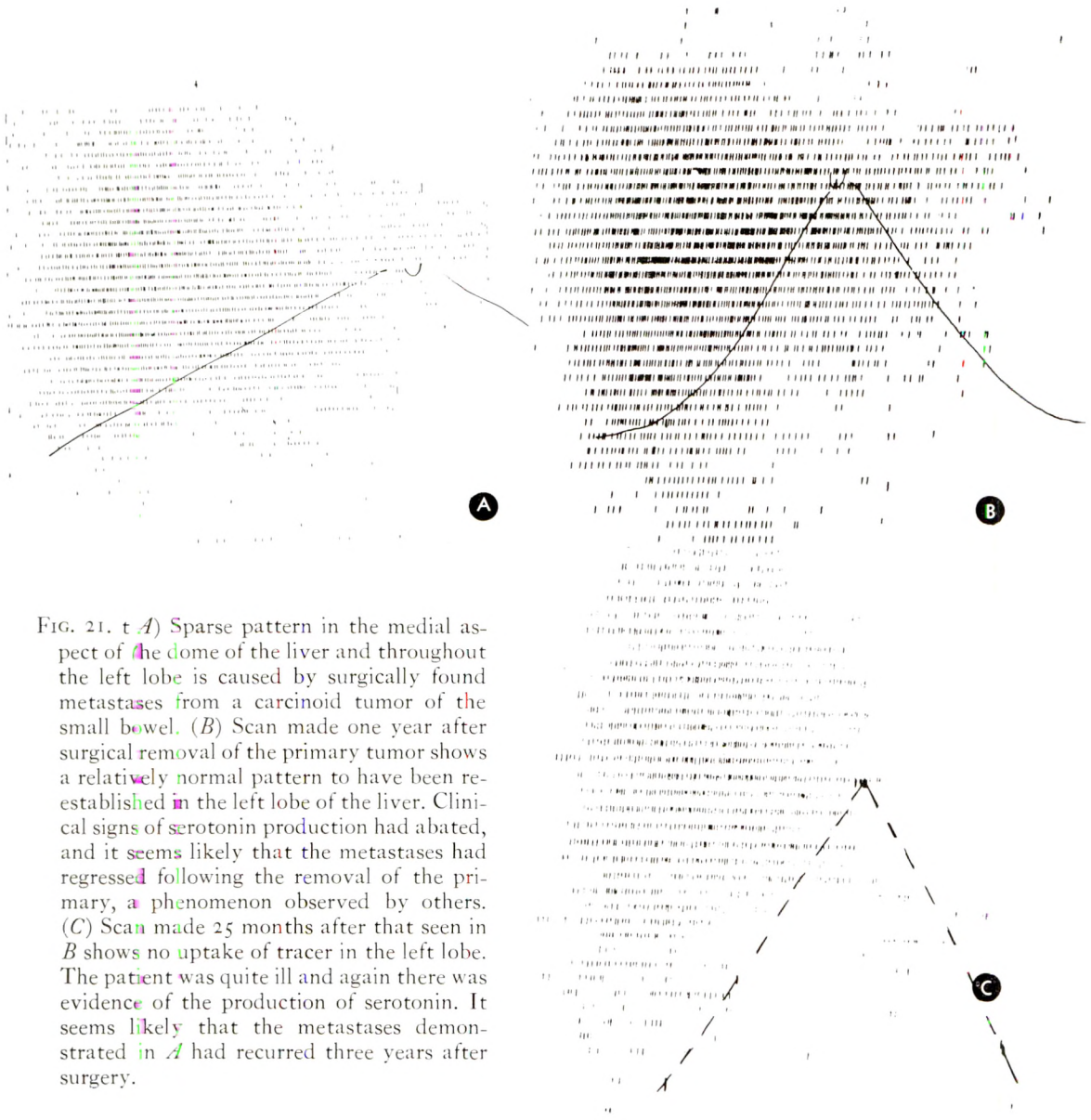


Fig. 21. t *A*) Sparse pattern in the medial aspect of the dome of the liver and throughout the left lobe is caused by surgically found metastases from a carcinoid tumor of the small bowel. (*B*) Scan made one year after surgical removal of the primary tumor shows a relatively normal pattern to have been reestablished in the left lobe of the liver. Clinical signs of serotonin production had abated, and it seems likely that the metastases had regressed following the removal of the primary, a phenomenon observed by others. (*C*) Scan made 25 months after that seen in *B* shows no uptake of tracer in the left lobe. The patient was quite ill and again there was evidence of the production of serotonin. It seems likely that the metastases demonstrated in *A* had recurred three years after surgery.

disease. Examination of the initial scan (Fig. 20 *A*) in retrospect suggests that the observer should have been more concerned with the discontinuities in the dome of the liver and along the superior margin of the left lobe, for they probably afforded preoperative evidence of metastases.

The case shown in Figure 21, *A–C* is another example of the use of the hepatoscan in following the progress of neoplastic disease in the liver, and a fascinating one. The patient was a sixty-six year old white woman who had had a carcinoid tumor of the small bowel removed recently. The findings of serotonin syndrome had been present. A group of metastases, identified at the operating table, created the defect in the dome and left lobe of the liver seen in Figure 21 *A*. One year later the patient returned for a follow-up scan. She had gained weight, ap-

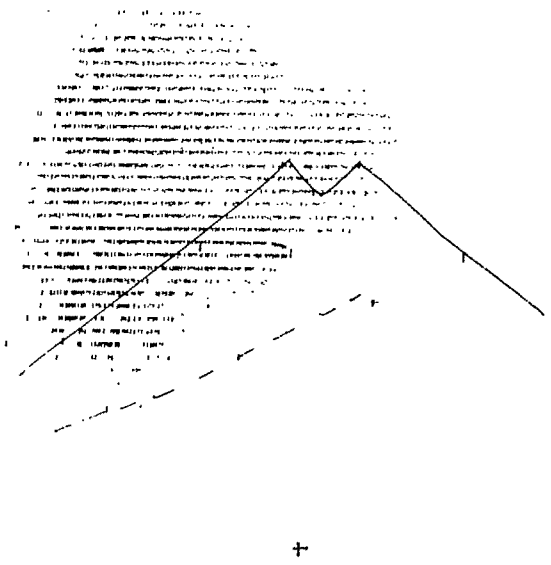


FIG. 22. Except for a few small defects in the midportion of the liver, the internal pattern is fairly uniform, but the apparent margin of the liver does not correspond with the edge of the mass (dotted line) palpable beneath the costal margin (solid line). At autopsy most of the liver was replaced by a hepatoma. The appearance of this scan was due to the presence of a rim of normal tissue extending over the tumor anteriorly (see text).

peared healthy, and the liver scan no longer showed the defects in the left lobe (Fig. 21 *B*). It seems likely that, as has been heretofore reported, the metastases of the carcinoid tumor within the liver had regressed after the removal of the primary neoplasm. Twenty-five months later, she returned, once again ill, and there was clinical evidence of the production of serotonin. A liver scan at that time (Fig. 21 *C*) now showed the left lobe of the liver to be truncated, no doubt due to the recurrence of the metastatic liver deposits.

Errors in scintillation scanning of the liver may result from the inability of available instruments to discriminate small space-occupying lesions, or even rather large ones, if they are buried deep within the substance of the liver. On several occasions we have scanned patients known to have multiple small liver abscesses or metastases and have been unable to identify them. Sometimes the anatomic nature of the space-occupying process itself makes interpretation difficult. In the case shown in Figure 22 there was a mass palpable in the right upper quadrant, with its margin represented by the dotted line. The scan shows only a few small defects but the pattern of the liver does not reach the edge of the mass. Such an appearance might be caused by a mass caudad to the liver, or a process occupying the inferior surface of the right lobe medially. At autopsy, however, a huge hepatoma had replaced much of the liver, and apparently only a thin rim of normal tissue was present over the anterior surface of this vast neoplasm. The true state of affairs would have been manifest had we scanned the patient in the lateral position, but time seldom permits us to obtain this additional projection. The use of a curved-path scanner[12] probably would have resulted in the detection of this lesion.

The dotted line in Figure 23 represents the margin of a huge, rounded, palpable mass which occupied much of the abdomen. The pattern of the liver itself is not especially remarkable, and it was thought that

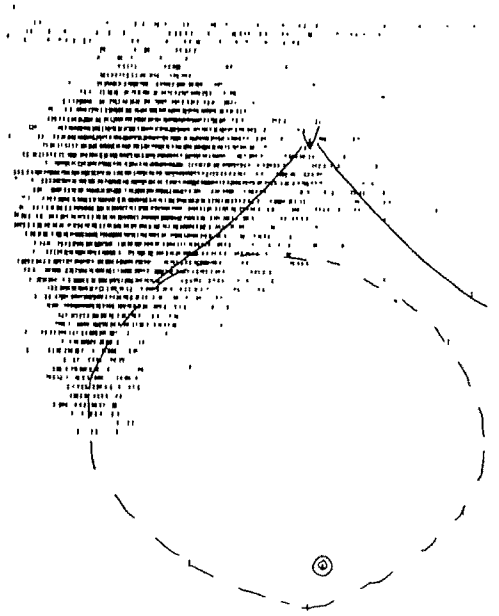


FIG. 23. Internal pattern is relatively normal, and so it was felt that the huge mass (dotted line) palpable well below the costal margin (solid line) in both upper quadrants was not of hepatic origin. At autopsy it was found that the mass arose from the under surface of the right lobe.

the mass in question was certainly not of hepatic origin. At autopsy, however, a large hepatoma arose from the under surface of the right lobe and projected into the abdomen, but occupied a very small volume within the substance of the liver itself.

Lastly, liver scanning, just as any other medical test, is subject to errors in interpretation such as that made in the case illustrated in Figure 20, *A* and *B*. Only extended experience will result in minimal errors of this sort.

SUMMARY AND CONCLUSIONS

1. Radioisotope scintillation scanning of the liver has been practiced in a number of institutions for over five years but its use is still not widespread. It may be performed reliably with most commercially available systems, if they are suitably adapted to this technique.[11] Patient radiation exposure is not sufficient to outweigh the medical evidence obtained.

2. Although liver scanning will give certain information about changes in size and shape of the liver, it is principally useful in estimating the position of the liver with respect to that of an apparently elevated right diaphragmatic leaf, as in subphrenic abscess formation, and in demonstrating the derangements of internal pattern produced by space-occupying lesions. It is a valuable test to employ in the study of patients who are thought to have, or who are being treated for, malignant neoplasms.

3. The procedure is subject to certain errors. These are due to the equipment with which it is performed, the distribution of disease within and about the liver, as well as to the human error of interpretation. With certain projected improvements in equipment and additional experience on the part of the observer, this important radioisotope test will become an accurate diagnostic technique.

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ESTIMATION OF LIVER SIZE BY THE MULTIPLE CUT OFF SCINTILLATION SCANNING TECHNIQUE*

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THE accurate determination of liver size is often important in the diagnosis and prognosis of liver disease. The clinical estimation of liver size, including the use of plain roentgenograms often gives erroneous results.[4,5,9,13]

Several investigators[2,7,9,13] have described more detailed roentgenographic and clinical methods for the volumetric determination of the liver. While these are more accurate than the routine clinical techniques, they have failed in the presence of various diseases of the chest and abdomen such as in pleural effusion and massive ascites which prevent the accurate location of the liver margin. The contrast visualization of the liver by means of portal venography while overcoming this difficulty is a formidable procedure[5] for this purpose alone.

With the successful outlining of the liver by scintillation scanning,[6] it was felt that this method would be useful in the volumetric estimation of liver size.

SCANNING TECHNIQUE

In the visualization of the liver by scintillation scanning, the materials used, colloidal Au^{198} and iodinated I^{131} rose bengal, are distributed evenly throughout the liver parenchyma. It follows, therefore, that the thicker areas of the liver will exhibit to the external counter higher counting rates than the thinner areas. In the multiple cut off technique of scintillation scanning, previously described in detail,[8] the setting of the cut off level ensures that only those areas of the liver exhibiting a higher counting rate than the level selected will be shown on the scan record. Thus, as the setting of the cut off is increased, the area of the scan record will be decreased so that ultimately only the thickest area of the liver will be visualized on the scanogram.

In the present series both colloidal Au^{198} and rose bengal I^{131} were used in doses ranging from 100 μc to 400 μc. A heavily shielded straight bore collimator of one half inch diameter and 3 inch length, as well as a focusing collimator (Picker Model 2107) was used. Counting rates varied from 1,500 to 5,000 counts per minute. Two or 3 cut off levels[8] were obtained on each patient. The peak counting rate over the liver in each patient was normalized to 100 per cent. The percentage cut off level then refers to the fraction of the peak count that must be exceeded to be recorded.

CALCULATION OF LIVER SIZE

As expected, appreciable differences in the size of each of the 3 scanograms were found (Fig. 1). Similarly, the degree of relative decrease in the size of each scanogram for the same cut off levels also differed, dependent upon the configuration of the liver. Therefore, in our method of determination of the liver size, the percentage cut off levels and the size of the scanograms obtained for the corresponding cut off levels were used as a function of liver size. Assuming the shape of the liver to be approximated by a truncated cone consisting of four small subsegments (Fig. 2), the following empirical formula was devised:

$$W = (V_1 + V_2 + V_3 + V_4)X \qquad (1)$$

In this formula W represents the weight of the liver. V_1, V_2, V_3, V_4 represent the volume of each subsegment respectively. X is the conversion factor used to relate the

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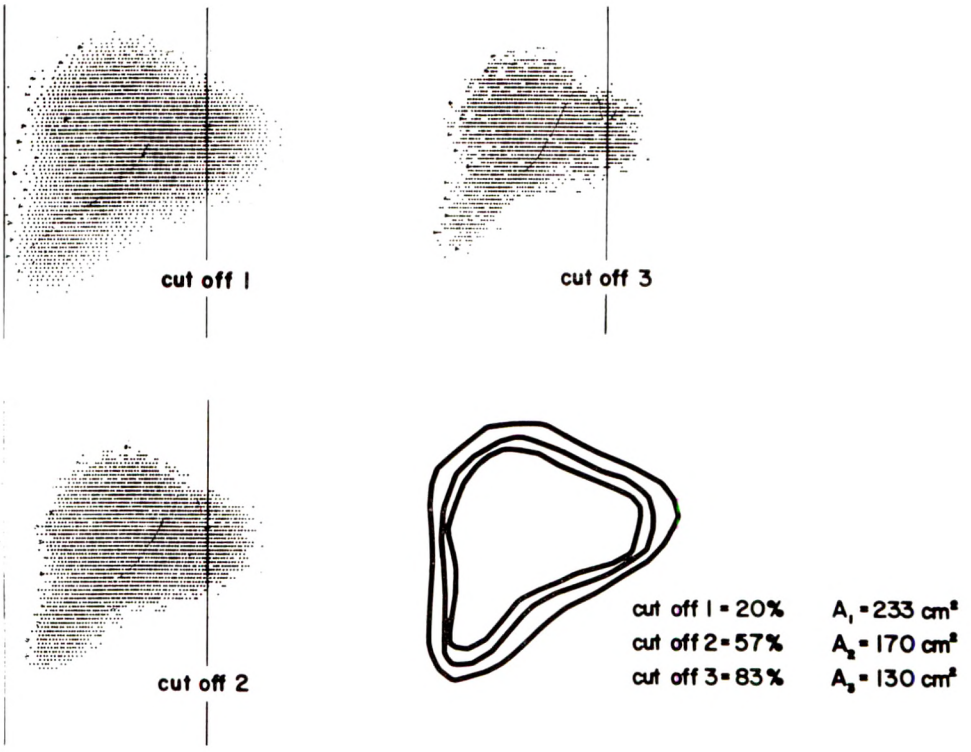


FIG. 1. Scanograms obtained at three different cut off levels showing decreasing size as the cut off level is increased. On the right are the superimposed contours of the scanograms with their measured areas and corresponding cut off levels. The weight of the liver was 1,700 grams.

measured volume of the truncated cone to the mass of the liver.

V_1, V_2, V_3, V_4 can be determined by the following formulas:

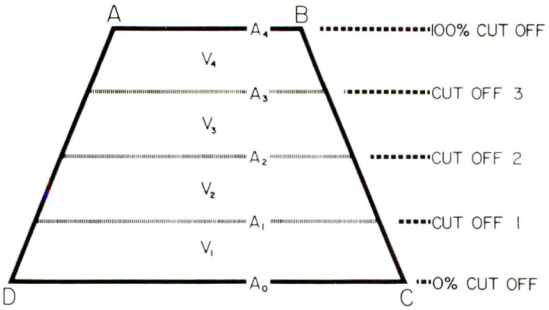


FIG. 2. ABCD truncated cone represents the approximated shape of the liver consisting of four subsegments (V_1, V_2, V_3, and V_4). A_1, A_2, A_3 are the areas of scanograms obtained at cut off 1, cut off 2 and cut off 3, respectively. A_0 and A_4 represent approximated values of scanograms which would have been obtained at zero and 100 per cent cut off levels.

$$V_1 = \frac{A_0 + A_1}{2} \cdot \text{cut off 1} \qquad (2)$$

$$V_2 = \frac{A_1 + A_2}{2} \cdot (\text{cut off 2} - \text{cut off 1}) \qquad (3)$$

$$V_3 = \frac{A_2 + A_3}{2} \cdot (\text{cut off 3} - \text{cut off 2}) \qquad (4)$$

$$V_4 = \frac{A_3 + A_4}{2} \cdot (1 - \text{cut off 3}) \qquad (5)$$

Here A_1, A_2, and A_3 are the actual areas of the scanograms measured with the planimeter.* Cut off 1, cut off 2, and cut off 3 are the known preset values of the cut off levels. Therefore, the only unknowns are A_0 and A_4, which correspond to the area of the scanograms, which would have been obtained if we used no cut off and 100 per cent cut off respectively. The zero and 100

* Keuffel & Esser Co., Compensating Polar Planimeter #4236M.

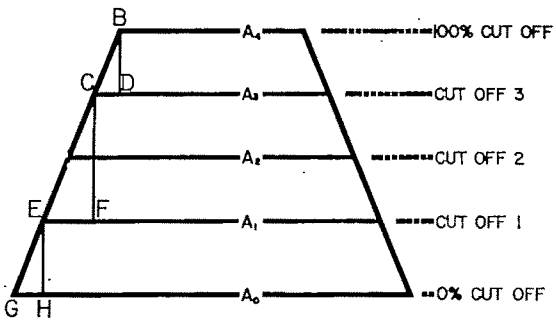


FIG. 3. The values of A_0 and A_4 are determined from the geometric relationships of BCD, CEF and EGH triangles, the known values of A_1, A_2, A_3 and their respective cut off values.

per cent cut off levels can be approximated by extrapolating the general contour of the liver as predicted by the slope of the line connecting the recorded cross sections and by using the following formulas obtained from the geometric relationships of BED, CEF, and EGH triangles (Fig. 3):

$$A_0 = \left[\sqrt{A_1} + \frac{\text{cut off 1}}{\text{cut off 3} - \text{cut off 1}} \cdot (\sqrt{A_1} - \sqrt{A_3})\right]^2 \quad (6)$$

$$A_4 = \left[\sqrt{A_3} + \frac{1 - \text{cut off 3}}{\text{cut off 3} - \text{cut off 1}} \cdot (\sqrt{A_1} - \sqrt{A_3})\right]^2 \quad (7)$$

The conversion factor (X) was determined experimentally from 48 patients in which the value of V_1, V_2, V_3 and V_4 was calculated from premortem scan records by using the above formulas 2, 3, 4, 5, 6, 7 and compared to the postmortem liver weights as calculated by equation 1.

RESULTS

The average value of X was found to be equal to 9.6 with a standard deviation of 1.3 (Table 1). Later, using this average value of X and equation 1, the weight of the liver was calculated in the above studied 48 cases. Table II shows the comparison of the calculated liver weights with the actual liver weight, as well as the percentage dif-

TABLE I

DETERMINATION OF THE AVERAGE VALUE OF X

Scan No.	N	X	X-X′	(X-X′)²
302	1	8.10	−1.53	2.35
303	2	9.65	+0.02	0.0004
337	3	7.40	−2.23	5.00
248	4	7.80	−1.83	3.35
237	5	11.50	+1.87	3.50
333	6	10.50	−0.87	0.76
236	7	9.50	−0.13	0.017
243	8	11.50	+1.87	3.50
341	9	10.50	+0.87	0.76
332	10	10.80	+1.17	1.38
238	11	10.20	+0.57	0.325
244	12	7.95	−1.68	2.84
240	13	10.30	+0.67	0.45
146	14	10.90	+1.27	1.62
276	15	8.75	−0.08	0.78
376	16	9.20	−0.43	0.186
246	17	11.20	+1.57	2.46
265	18	9.80	+0.17	0.029
330	19	9.40	−0.23	0.053
481	20	9.22	−0.41	0.168
399	21	7.65	−1.98	3.94
197	22	8.40	−1.23	1.52
198	23	11.70	+2.07	4.20
229	24	9.80	+0.17	0.029
155	25	8.60	−1.03	1.06
184	26	10.50	+0.87	0.76
145	27	12.70	+3.07	9.45
165	28	9.35	−0.28	0.079
164	29	10.70	+1.07	1.16
159	30	9.00	−0.63	0.40
191	31	9.30	−0.33	0.10
174	32	10.00	+0.37	0.137
225	33	9.30	−0.33	0.10
140	34	7.85	−1.78	3.18
196	35	8.55	−1.08	1.165
175	36	9.80	+0.17	0.029
118	37	7.00	−2.63	6.95
176	38	11.60	+1.97	3.90
199	39	10.00	+0.37	0.138
206	40	9.45	−0.18	0.032
265	41	10.00	−0.37	0.137
167	42	11.60	+1.97	3.90
223	43	10.80	+1.17	1.40
182	44	10.70	+1.07	1.16
192	45	9.10	−0.53	0.28
190	46	10.40	+0.77	0.595
181	47	7.20	−2.43	5.90
256	48	7.20	−2.43	5.90
	48	462.42	−0.56	86.10

N = Number of cases studied.
X = The value of conversion factor on each case.
X′ = The average value of X.
Average value of X = 9.63. Standard deviation = 1.35.

TABLE II

COMPARISON OF THE CALCULATED WEIGHT OF THE LIVER WITH THE ACTUAL LIVER WEIGHT

Scan No.	Liver Findings	Calculated Weight	Actual Weight	Δ W %
155	Metastatic Carcinoma	2,000	1,790	+11.7
184	Fatty Liver	1,400	1,520	− 8.0
145	Metastatic Carcinoma	1,360	1,790	−24.0
165	Primary Carcinoma	2,100	2,050	+ 2.9
164	Passive Hyperemia	1,150	1,275	− 9.8
159	Metastatic Carcinoma	1,030	960	+ 7.3
191	Metastatic Carcinoma Passive Hyperemia	2,340	2,260	+ 3.5
174	Normal	1,380	1,425	− 3.2
225	Normal	1,195	1,150	+ 3.9
140	Tumor Invasion, Right Lobe	1,600	1,300	+23.0
196	Metastatic Carcinoma	1,660	1,475	+12.5
175	Passive Hyperemia	1,300	1,325	− 1.9
118	Normal	1,470	1,075	+36.8
176	Passive Hyperemia	1,590	1,910	−16.7
199	Normal	1,460	1,525	− 4.2
206	Cholangitis	1,145	1,125	+ 1.8
264	Normal	1,360	1,400	− 2.9
223	Metastatic Carcinoma, Massive	1,210	1,350	−10.4
182	Normal	1,010	1,125	−10.2
192	Passive Hyperemia	1,460	1,390	+ 5.0
190	Passive Hyperemia; Biliary Cirrhosis	1,860	2,050	− 4.4
181	Passive Hyperemia	2,070	1,540	+34.4
256	Fatty Liver	2,500	1,870	+33.6
302	Leukemic Infiltration	1,730	1,450	+19.3
303	Metastatic Carcinoma	3,015	3,030	− 0.5
337	Atrophy	675	520	+11.2
248	Normal	1,815	1,470	+23.4
237	Fatty Liver	1,350	1,620	−16.7
333	Normal	1,125	1,220	− 7.9
236	Laennec's Cirrhosis	1,180	1,160	+ 1.7
243	Normal	1,155	1,375	−16.0
341	Lymphoblastoma	2,580	2,825	− 8.6
332	Fatty Liver	2,000	2,250	−11.1
238	Passive Hyperemia	1,735	1,840	− 5.7
244	Leukemic Infiltration	2,190	1,800	+21.6
240	Normal	1,135	1,220	− 6.9
146	Fatty Liver	1,390	1,570	−11.5
276	Metastatic Sarcoma	1,595	1,450	−10.0
376	Hodgkin's Disease	2,142	2,050	+ 4.5
246	Metastatic Carcinoma	1,150	1,455	−21.0
265	Normal	1,377	1,400	− 1.6
330	Normal	1,580	1,530	+ 3.3
481	Fatty Liver; Metastatic Carcinoma	1,775	1,700	+ 4.4
399	Fatty Liver	1,910	1,520	+25.6
197	Metastatic Carcinoma Infarct	1,890	1,650	+14.5
198	Metastatic Carcinoma	1,720	2,090	−17.6
229	Normal	1,580	1,600	− 1.3
167	Passive Hyperemia	775	930	−16.7

Total number cases studied = 48. Average deviation = 11.56 per cent.

(*Continued at the right*)

ferences. The average deviation was found to be equal to 11.6 per cent.

DISCUSSION

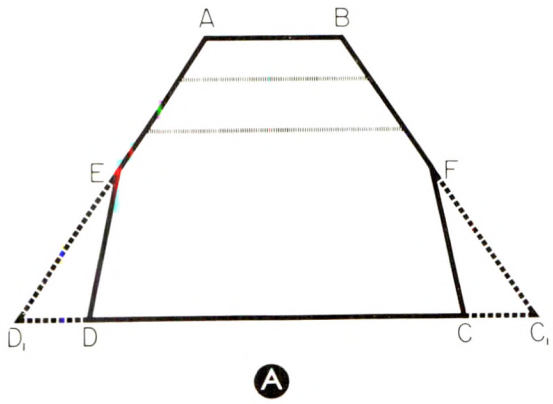
The errors in liver weight estimations are attributed primarily to the uncertainty in the prediction of the extrapolated contours of the liver. While in most cases this was due to the selection of cut off levels which were not optimal for liver size determinations, in some it was complicated by abnormal configuration.

As shown in Figure 4, *A* and *B*, livers whose radioactive profile[8] exhibit a wide waist and narrow upper end and where only 2 very high or very low cut offs have been used, volume estimates were high. The reverse of this, low volume estimation, was found in livers whose radioactive profiles exhibit a narrow waist and when only two very high or very low cut offs have been used (Fig. 5, *A* and *B*).

The errors in estimations can be improved either by using two cut offs in better separation or by adding a third cut off. While theoretically a zero cut off would be advantageous, this is not feasible with our scanner because of high body background which makes the actual extent of the liver at the zero level more arbitrary than a predicted extrapolation. Similarly, recording of cut offs greater than 80 per cent have exhibited too great a random fluctuation so as to make accurate area measurements difficult.

The present accuracy of 11.6 per cent average deviation has been obtained from scans made at cut off levels chosen for visualization of defects rather than for liver size determination. Also, over half of the scans were obtained with only two cut

off levels rather than the preferred three level technique. Therefore, it is believed, that the accuracy of this method would be considerably improved if used specifically for liver size determination. While these results were obtained using both straight bore and focusing collimators which showed no measurable difference in our series, it must be emphasized that the conversion value of X is directly applicable only to the described counting system.



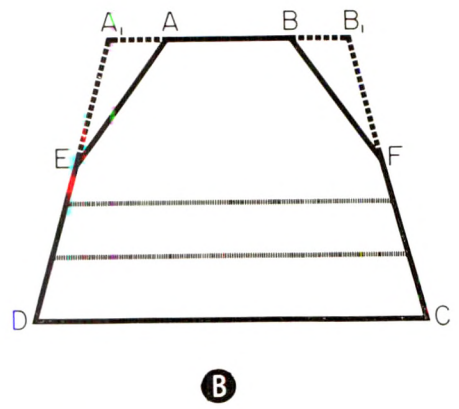
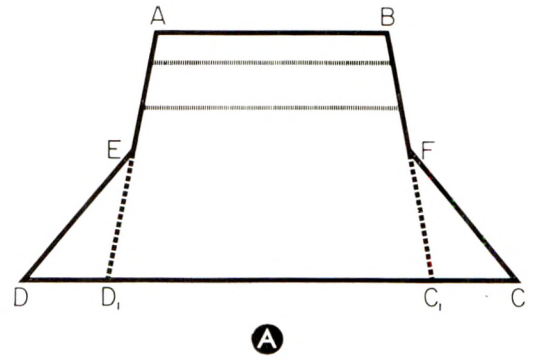


Fig. 4. (*A*) ABFCDE = actual size of the liver. ABC_1D_1 = calculated size of the liver. Dotted transverse lines represent areas of scanograms obtained at two high cut off levels. EDD_1 and FCC_1 represent volumes included by extrapolation, resulting in a false high estimate. (*B*) ABFCDE = actual size of the liver. A_1B_1CD = calculated size of the liver. Dotted transverse lines represent the areas of scanograms obtained at two low cut off levels. A_1AE and BB_1F represent volumes included by extrapolation, resulting in a false high estimate.



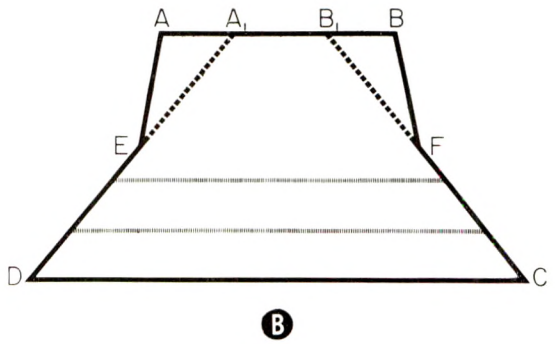


Fig. 5. (*A*) ABFCDE = actual size of the liver. ABC_1D_1 = calculated size of the liver. Transverse dotted lines represent areas of the scanograms obtained at two high cut off levels. ED_1D and FCC_1 are part of the true volume of the liver which are not accounted for by the extrapolation, resulting in a false low estimate. (*B*) ABFCDE = actual size of the liver. A_1B_1CD = calculated size of the liver. Dotted transverse lines represent areas of the scanograms obtained at two low cut off levels. AA_1E and B_1BF are part of the true volume of the liver which are not accounted for by the extrapolation, resulting in a false low estimate.

While large deviations are not expected, this value would require individual determinations for use with other counting systems.

This technique provides an entirely objective method that can be obtained from specific parameters rather than from subjective data derived from the clinical techniques. In the follow-up cases where comparative estimates are required, this method has proved extremely valuable. In patients with ascites this technique has been the only system that has been found

TABLE III

DETERMINATION OF THE DENSITY OF THE LIVER

Case No.	Liver Findings	Density
1	Metastatic Carcinoma, Massive	1.06
2	Normal	1.04
3	Passive Hyperemia, Central Necrosis	1.04
4	Normal	1.05
5	Passive Hyperemia, Central Necrosis, Fatty Change	1.02
6	Atrophy	1.04
7	Passive Hyperemia	1.05
8	Passive Hyperemia	1.04
9	Passive Hyperemia	1.04
10	Metastatic Carcinoma, Massive	1.04
11	Fatty Liver	1.04
12	Fatty Liver	1.04
13	Metastatic Carcinoma	1.05
14	Passive Hyperemia	1.05
15	Normal	1.04
16	Fatty Liver	1.05
17	Metastatic Carcinoma	1.04
18	Metastatic Carcinoma; Fatty Liver	1.04
19	Metastatic Carcinoma	1.05
20	Normal	1.05
21	Normal	1.03
22	Laennec's Cirrhosis	1.03
23	Metastatic Carcinoma; Fatty Liver	1.04
24	Passive Hyperemia	1.04
25	Metastatic Carcinoma, Massive	1.05
26	Laennec's Cirrhosis	1.04
27	Passive Hyperemia	1.05
28	Passive Hyperemia; Cardiac Cirrhosis	1.04
29	Normal	1.05
30	Remote Infarct	1.05
31	Atrophy; Fatty Liver	1.03
32	Passive Hyperemia	1.05
33	Passive Hyperemia	1.03
34	Normal	1.03
35	Fatty Liver	1.04
36	Fatty Liver	1.04
37	Fatty Liver	1.03

Average density of the liver = 1.04. Standard deviation = .01.

satisfactory in the evaluation of size of normal and cirrhotic livers. In patients with tumors of the periphery of the liver, although the absolute values of the liver mass have been uniformly low, comparative results have still proved useful.

In an additional study the density of the liver was determined in 37 autopsy cases and was found to be equal to 1.04 (Table III). Since no significant variations were seen in the densities of different cases studied, the volume of the liver can be also determined by dividing the calculated weight of the liver by 1.04, or in the formula 1 by using the value of X equal to 9.24.

CONCLUSIONS

An objective method of determining liver size, even in cases where the margins are obscured clinically and roentgenographically, using the multiple cut off scintillation scanning technique has been described. Comparison with postmortem data indicates an accuracy of ±11.6 per cent. Methods of improving this accuracy are discussed.

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A COMPARISON OF I^{131} ROSE BENGAL AND COLLOIDAL AU^{198} IN LIVER SCANNING*

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THE general use of I^{131} rose bengal for visualizing the structure of the liver has been accompanied by many problems inherent in utilizing a material which is both accumulated and released at rates varying considerably from patient to patient. These difficulties have been threefold. First, a continually changing counting rate cannot be readily adapted to a set accentuation technique, since a setting at the early part of the scan may not be optimum at the end. Secondly, the concentration in the gallbladder is recorded as an area of activity much greater than the surrounding liver tissue. The third problem involves the release of the material into the intestine before the scan is completed, thus obscuring the limits of the liver and presenting artifacts indistinguishable from normal liver tissue.

To be as useful in liver scanning as I^{131} rose bengal, the distribution of colloidal Au^{198} in the Kupffer cells must give approximately the same radioactive distribution as the accumulation of radioactive rose bengal by the parenchymal cells. This was in effect established by Root *et al.*,[14] who by means of gross radioautography of the liver in various pathologic conditions demonstrated the distribution of colloidal gold in reasonably normal liver parenchyma to be quite uniform, with a fine pattern of increased concentration related to lobular architecture. They further state that compared with isotope distributions, in general, this is one of the most homogeneous they have seen.

This uniform distribution is not surprising if one considers that about 40 per cent of the cells in the liver are not parenchymal cells and that a large proportion of the nonparenchymal cells are reticuloendothelial cells capable of taking up the radioactive colloidal gold.[5] Similar uniformity of distribution of rose bengal has been reported by Glaser *et al.*[8] and Bocci[2] by means of microscopic radioautography. In the latter study, radioactive rose bengal was identified in both parenchymal and Kupffer cells 20 minutes following injection.

Although both materials have been used by many investigators for scintillation scanning,[1,4,6,7,10,13] the present investigation has been concerned with the extension of the microscopic uniformity to the macroscopic resolution obtained by the scintillation scanner. If this is satisfactory then the variation in the gross anatomic distribution of these 2 materials may be compared with regard to problems of scanning itself.

METHOD

To compare the usefulness of I^{131} rose bengal and colloidal Au^{198} in liver scanning, 45 patients with varying clinical conditions were studied. These patients, with only a few exceptions, were scanned first with colloidal Au^{198} and then, after an appropriate delay to allow for a suitable decrease in residual activity, scanned again with I^{131} rose bengal. All scanning parameters, such as maximum counting rate, traverse speed, cut off levels, and dot factors were kept as similar as possible. Only those patients were included in whom the period between scans was short or whose clinical condition would permit comparison with longer delays.

The dosage of both the colloidal Au^{198}

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and I^{131} rose bengal ranged from 300 to 500 μc which was sufficient to achieve a peak counting rate of approximately 4,000 counts per minute over the right lobe of the liver. The detector used in this study consisted of a heavily shielded $\frac{3}{4} \times \frac{3}{4}$ inch thallium activated NaI crystal with a 3 inch straight bore collimator of $\frac{1}{2}$ inch diameter. The scanning speed was 8 inches per minute. In general, 3 dot scans were simultaneously recorded with cut off levels of approximately 25 per cent, 60 per cent, and 75 per cent of the peak counting rate. The exact setting of the cut off levels was determined by our routine survey linear profiles.[11] A $\frac{1}{2}$ second time constant was employed in the cut off circuit.

To establish a comparison of the macroscopic distribution of colloidal Au^{198} and I^{131} rose bengal, 2 moribund patients with Laennec's cirrhosis were injected shortly before death, one with 500 μc of I^{131} rose bengal and the other with 500 μc of colloidal Au^{198}. At autopsy sections of the liver were obtained for gross radioautographs.[3]

RESULTS

Autoradiographs and photographs of the livers of the 2 patients with Laennec's cirrhosis are shown in Figure 1, *a–d*. Figure 1*a* shows the liver autoradiograph of the patient injected with I^{131} rose bengal. The remaining liver lobules are clearly delineated from the extensive scarring characteristic of this disease. Figure 1*c* shows a photograph of the whole organ demonstrating the typical nodularity of advanced Laennec's cirrhosis. Figure 1*b* shows the liver autoradiograph of the patient injected with colloidal Au^{198}. Again, the distribution of the radioactive material is uniform throughout the liver lobules. The triangle of decreased activity at the top left is due to an infarct of this area, which can be seen

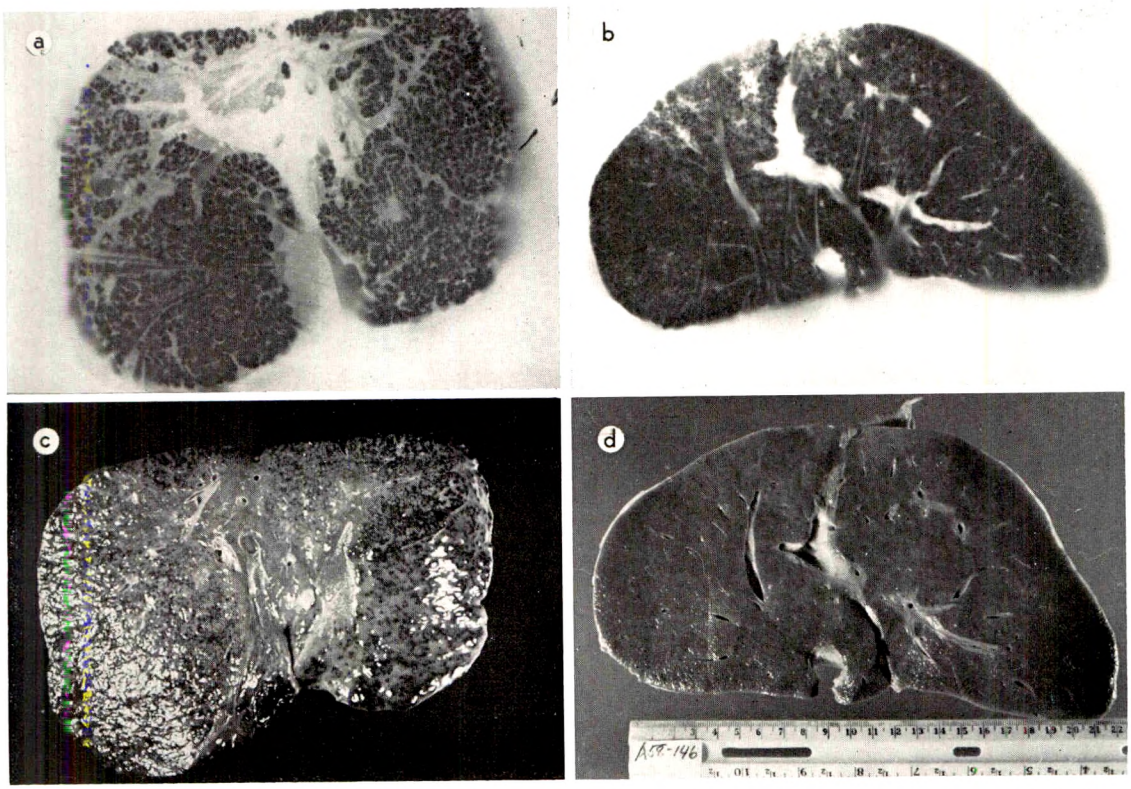


FIG. 1. Autoradiographs and photographs of livers in patients with Laennec's cirrhosis. (*a* and *c*) Patient was injected shortly before death with 500 μc of I^{131} rose bengal. (*b* and *d*) Patient was injected with 500 μc of colloidal Au^{198}.

faintly outlined on the photograph of this liver slice (Fig. 1*d*). It is apparent that while both patients had Laennec's cirrhosis, the former liver (Fig. 1, *a* and *c*) shows more extensive scarring. In both, however, radioactivity is evenly distributed throughout the remaining normal liver lobules.

To demonstrate the uniformity of distribution of the I^{131} rose bengal and colloidal Au^{198}, a patient without evident liver disease was scanned with both materials. Comparative scans (Fig. 2, *a* and *b*) at identical cut off levels demonstrate similar radioactive distribution except for a slight increase in radioactivity over the gallbladder area in the I^{131} rose bengal scan.

Comparable scans, obtained with both materials in a patient with compensated Laennec's cirrhosis, are shown in Figure 3, *a–d*. Both show the liver to be enlarged with multiple small "defects" in the left lobe at the higher cut off level. These "defects" were statistically significant.

Similar agreement has not always been observed. Figure 4*a* represents a liver scan obtained with I^{131} rose bengal in an 88 year old patient with a history of partial resection of the liver and cholecystectomy. On physical examination the liver was described as irregular and enlarged with the question of metastatic disease. The I^{131} rose bengal scan shows an odd-shaped liver in which the lower pole of the right lobe appears absent. There is an area of irregular distribution in the lower right of the scan having the appearance of a clearly defined "defect" surrounded by normal liver tissue. The second hepatoscan (Fig. 4*b*), performed 2 weeks later with colloidal Au^{198}, shows a moderately enlarged liver with no significant defects. By superimposition, it can be demonstrated that the defect described in the I^{131} rose bengal scan is simply an artifact located outside the liver, and is due to the excretion of I^{131} rose bengal into the duodenum.

The patient whose scans are shown in Figure 5, *a* and *b*, was scanned first with I^{131} rose bengal and 1 week later with colloidal Au^{198}. Both scans show an enlarged liver with multiple large defects due to a metastatic carcinoma proven by postmortem examination. In the colloidal Au^{198} scan (Fig. 5*b*), an area of concentration of activity is seen below the right lobe. Counts over this area were almost twice as high as the peak activity within the liver. Twenty-four hours later this activity had dropped twelve-fold while the counts in the liver had decreased only 25 per cent. It is evident that this "mass" represented an artifact due to excreted I^{131} rose bengal in the right colon from the scan performed 1 week previously.

Figure 6*a* represents a normal liver scan obtained after the administration of radioactive gold. A comparison of the dot pattern with a series of linear profiles recorded simultaneously[9] shows an area of decreased activity in the inferior margin of the liver.

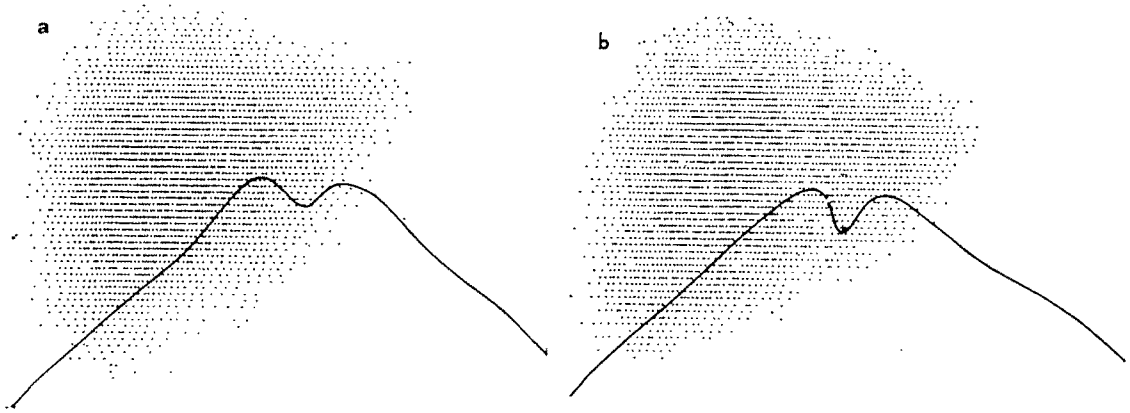


FIG. 2. Liver scans of patient injected (*a*) with I^{131} rose bengal and (*b*) colloidal Au^{198}.

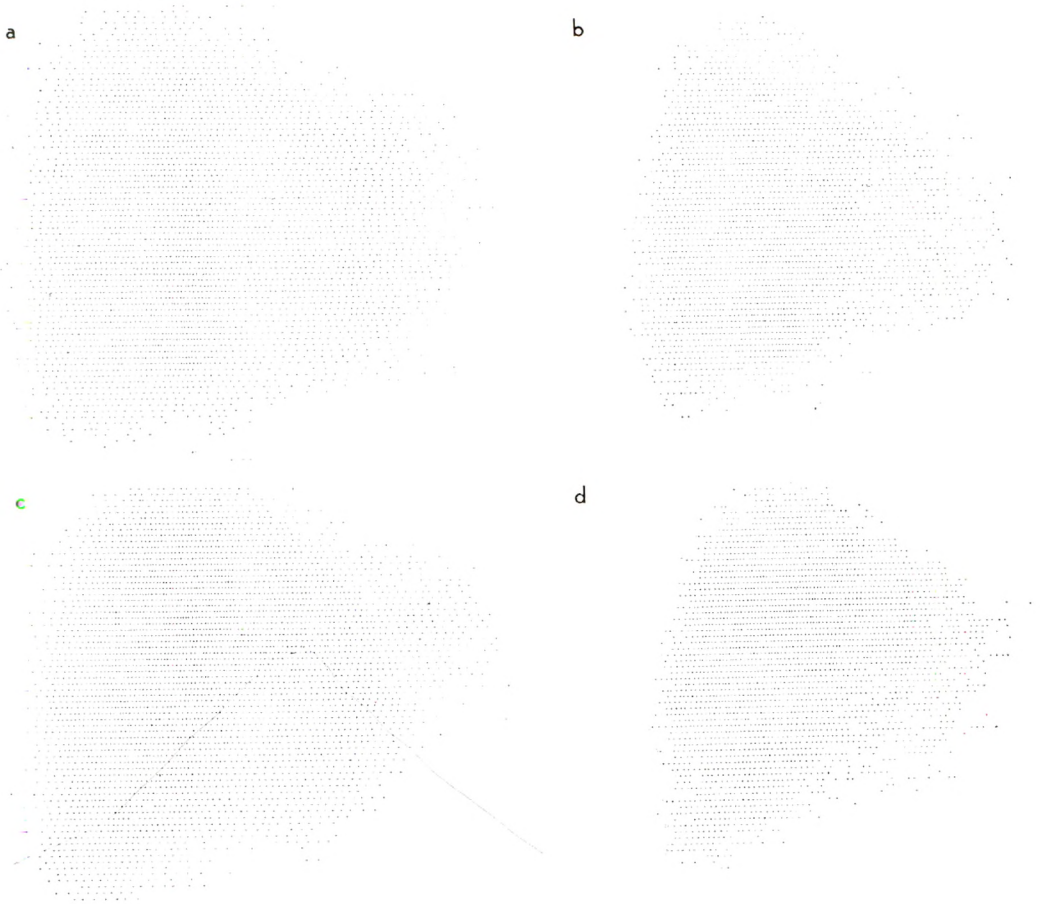


FIG. 3. Comparable liver scans in a patient with compensated Laennec's cirrhosis. (*a*) I^{131} rose bengal scan at 25 per cent cut off level and (*b*) at 60 per cent cut off level. (*c*) Colloidal Au^{198} scan at 25 per cent cut off level and (*d*) at 60 per cent cut off level. Note additional defects at the top of the I^{131} rose bengal scan at the 60 per cent cut off level.

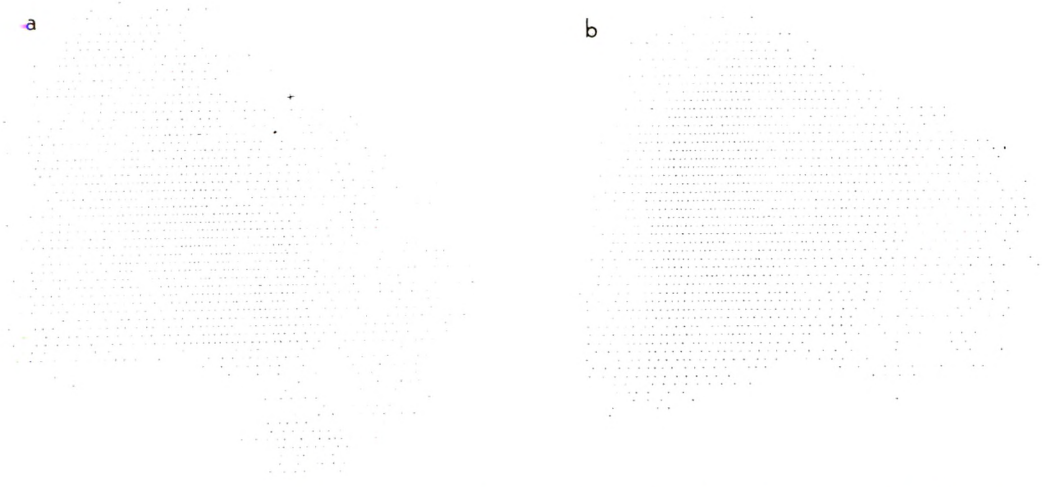


FIG. 4. (*a*) I^{131} rose bengal scan showing artifact due to the excretion of rose bengal into the duodenum. (*b*) Colloidal Au^{198} scan showing limits of the liver.

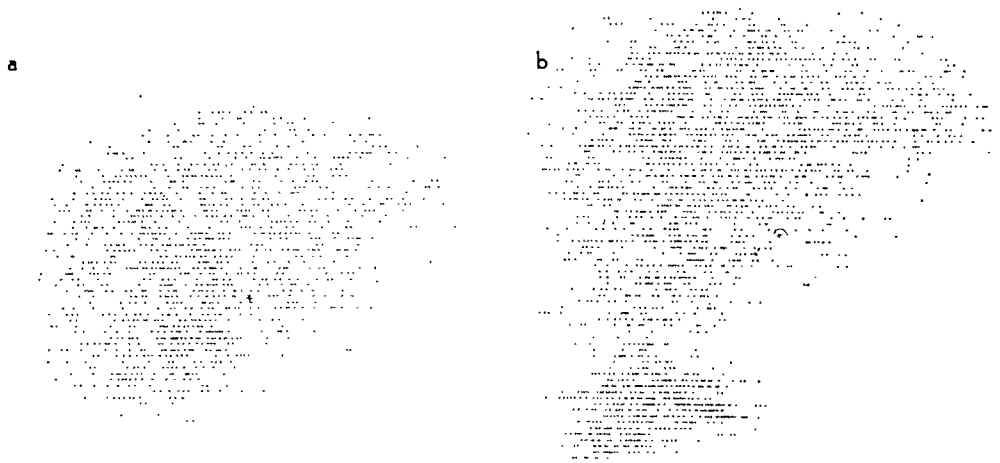


FIG. 5. (*a*) I^{131} rose bengal scan on a patient with metastatic disease showing multiple defects in radioactive distribution. (*b*) Colloidal Au^{198} scan obtained one week later showing persistence of I^{131} rose bengal "artifact" in the colon.

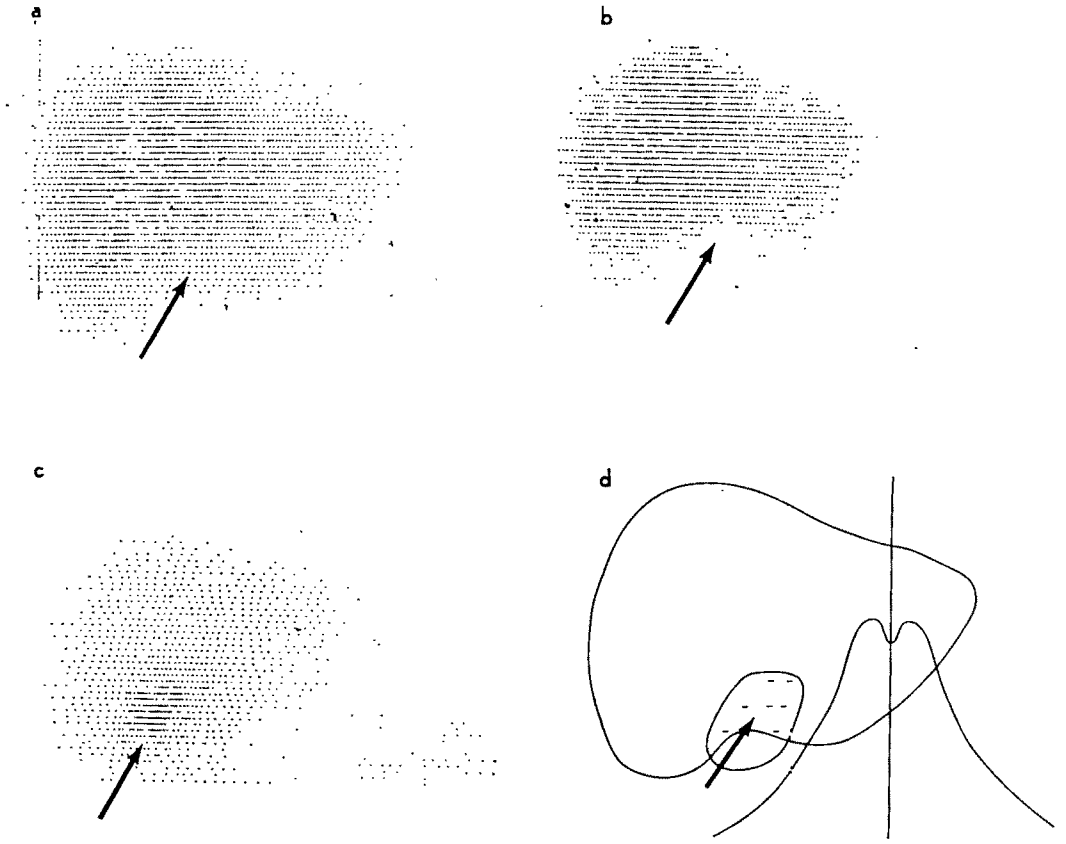


FIG. 6. (*a* and *b*) Colloidal Au^{198} scans showing area of decreased activity in the region of the gallbladder as seen (*c*) on the I^{131} rose bengal scan. (*d*) Schematic drawing showing superimposition of the area of decreased activity in the gold scan and the area of increased activity in the I^{131} rose bengal scan.

In a hepatoscan obtained at a higher cut off level (Fig. 6*b*), this area appears like a deep indentation. In the rose bengal scan (Fig. 6*c*) performed 2 weeks later, an area of increased activity was found in the same location as described above in the gold scans. It is evident that the same anatomic feature, the gallbladder, may be represented differently depending on the type of deposition of the radioactive material used. In this case, the impression of the gallbladder on the posterior surface of the liver, resulting in a thinner mass of tissue, is represented in the Au^{198} scan as an area of decreased activity. The rose bengal scan shows the gallbladder as an area of increased activity due to the selective concentration of the radioactive dye in that organ. Figure 6*d* is a schematic drawing representing the outline of the liver as shown in Figure 6*a*. The dashed lines represent the points of maximum drop in activity demonstrated by the profiles obtained during the Au^{198} scan. The outline of the gallbladder, as demonstrated by the rose bengal scan, has been also drawn in the same figure to show the anatomic correlation between the area of decreased activity in the Au^{198} scan and the area of increased activity in the rose bengal scan. A common finding is the artifact under the left costal margin due to rose bengal released into the intestine.

Another case of differential deposition is illustrated in Figure 7, *a* and *b*. When this patient was scanned with radioactive gold, the scan showed two distinct areas of deposition: the liver, which appears enlarged in its vertical diameter, and what most probably represents the spleen. Since rose bengal has no specific affinity for the cells of the reticuloendothelial system, no deposition is expected in the spleen. Therefore, the scan of the hypochondria after administration of I^{131} rose bengal has shown only the liver (Fig. 7*b*) plus the release of this material into the proximal small bowel.

The preceding example showed a clear-cut separation between liver and spleen; therefore, permitting a good delimitation of the two organs. This is not always the case. Figure 8*a* illustrates a gold scan on a patient with known metastatic disease. The radioactivity extends well beyond the midline. There is a deep indentation in the right inferior margin of the scan which suggests the possibility of a large space-occupying lesion in the left lobe of the liver. An I^{131} rose bengal scan performed 14 days later showed a moderately enlarged liver of normal shape with no suggestion of defects.

DISCUSSION

Although both of these materials show the same uniform distribution in normal liver tissue, it is apparent that there are advantages and disadvantages of each.

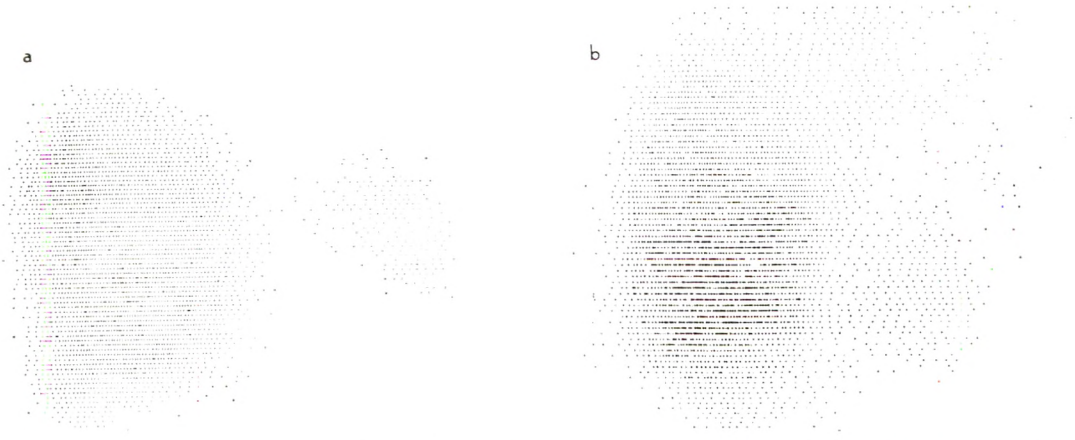


Fig. 7. (*a*) Colloidal Au^{198} scan showing radioactivity over the area of the spleen. (*b*) I^{131} rose bengal scan showing no activity in the region of the spleen. Note artifacts due to intestinal release.

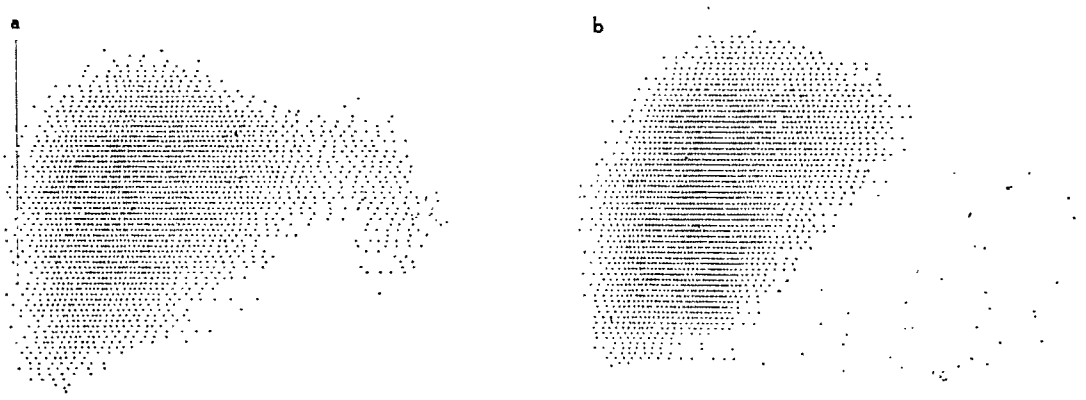


FIG. 8. (*a*) Colloidal Au^{198} scan showing radioactivity extending well beyond midline. (*b*) I^{131} rose bengal scan indicates that the radioactivity on the far right in the gold scan represented the spleen.

At Highland View Hospital we have been increasingly concerned with the diagnosis of liver disease with minimal morphologic alterations resulting in counting rate deviations in the range of 20 per cent. These deviations must be differentiated from such normal variations as engorged vessels, which may result in counting rate changes of this magnitude.

In view of this objective, we have relied considerably on accentuation techniques (cut off) which require stability of counting rate. With the fast excretion of I^{131} rose bengal, a continuously decreasing counting rate is encountered as the scan progresses. This results in a relative increase in cut off level and the production of artificial "defects" by the end of the scan.

The rose bengal scan of Figure 3*b*, obtained, as is customary, by moving the detector from the inferior to the superior margin of the liver to minimize bowel artifacts, shows small artificial "defects" at the top, which are not present in the scan obtained with colloidal Au^{198}, a more stable material (Fig. 3*d*).

In addition, while the artifacts in the I^{131} rose bengal scans, due to its release into the intestine, may not be too disconcerting as our experience increases, a substance which for practical purposes is selectively accumulated and retained in the liver is preferred.

In livers with severely impaired function and slow uptake, one additional disadvantage of I^{131} rose bengal occurs when equilibrium is not reached until considerable activity has been released to the intestine. If, in order to overcome this difficulty, the scan is started before the peak of activity is reached, an undesirable change in counting rate throughout the procedure necessarily results.

Our attempts to appreciably decrease the turnover rate of I^{131} rose bengal by the liver with the administration of 100 mg. of inactive rose bengal have been unsatisfactory. Working with rabbits, Koch-Weser *et al.*[9] have indicated that 20 mg. per kg. of body weight of inactive rose bengal is required to substantially decrease the turnover of this substance. If the same ratio applies to man, doses of approximately 1.4 gm. would be necessary.

Any accentuation technique requires the accurate pre-setting of the scanning parameters. Despite the most careful efforts, it is frequently desirable to repeat a scan with different settings. Radioactive gold is especially useful in these cases since its retention by the liver permits rescanning within 24 hours without reinjection.

With impairment of portal circulation, colloidal Au^{198} permits visualization of the spleen. This, in itself a significant clinical fact, will sometimes result in problems of interpretation. In these cases, clarification is readily achieved by rescanning with I^{131} rose bengal.

The primary disadvantage of colloidal Au^{198} is the higher dose exposure. Quimby[12] has recently calculated the dose to the liver from 150 μc of Au^{198} as 5.7 rads. This is identical with the original estimates of Friedell *et al.*[7] of 8 rads from a 210 μc dose. This exceeds the recommended level. Therefore, it is the concern of the physician to weigh the importance of the potential benefit against the hazards involved.

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CONTRAST ROENTGENOGRAPHY VERSUS RADIOISOTOPE SCANNING OF LIVER AND SPLEEN*

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AS A result of ten years of research, we have solved the problem of hepatosplenography in the rat without the use of relatively large quantities of radioactive elements or iodine. The use of small quantities of radioactive elements and scanning is rapidly replacing the radiopaque method in clinical diagnosis.[8,11] However, we feel that our work is not entirely lost as our technique in making nontoxic sols of tantalum can be applied to the making of sols of Ta^{173} with half-life of 3.7 days or other isotopes of tantalum or other elements as substitutes for Au^{198}.

TECHNIQUE AND RESULTS WITH TANTALUM PENTOXIDE SOLS

(1) Colloidal preparations of tantalum pentoxide tend to be gels rather than sols. Sorantin and Jantsch[9] were able to prevent gel formation by a rapid shearing action during hydrolysis of dilute solutions of tantalum pentachloride. The resulting dilute sol could be distilled *in vacuo* to 7 per cent tantalum pentoxide concentration, but no further without forming a gel. We found such sols lethal to rats. With modifications of the method of Sorantin and Jantsch, we prepared 7 per cent sols as follows: 5 ml. of 40 per cent tantalum pentachloride in absolute ethyl alcohol was added to a small amount of distilled water in a 200 ml. beaker. Twenty-five milliliters of 10 per cent dextrin and enough distilled water to make a total volume of 200 ml. were added. This was heated to 70° C. and transferred to a heated Pyrex Waring blender or Virtis homogenizer fitted with a metal thermometer and rotated at 1,500 or 4,500 r.p.m. until the mixture became reddish by transmitted light. The rotation had to be stopped before the temperature reached 80° C. at which point the sol changed to a gel. The sol was poured into a vacuum still immersed in a water bath of 30° C. The still was evacuated with a high vacuum pump which was protected from water vapor by passage of the evacuate through a flask immersed in ice and salt. To prevent bumping, the still was fitted with a capillary air inlet and this air was passed through anhydrous calcium chloride and cotton. When the concentration of the sol reached about 7 per cent Ta_2O_5, it was removed from the still and dialyzed 48 hours against a large volume of polyvinylpyrrolidone (PVP) in distilled water, used as a plasma substitute. Equal parts of sol and glycerol were mixed and neutralized with NaOH using Hydrion paper until sufficient electrolyte was added to allow a glass electrode to operate. The sol was boiled to oleate the Ta_2O_5. One milliliter was injected into a 250 gm. rat. At least two injections were necessary to opacify the liver and spleen. This was repeated successfully in more than 50 rats (Fig. 1).

(2) In order to obtain tantalum sols in pure glycerol, dry oleated Ta_2O_5 of less than 1 μ. size was ground with pure glycerol in a colloidal mill and placed in test tubes which were kept vertical and protected from temperature changes which produce convection currents over night. The upper portion was used to inject 50 rats of 250 gm. body weight in 1 ml. quantities. Again it was found necessary to make two injections in order to opacify the liver and spleen. Larger quantities could not be used owing to the toxicity of glycerol.

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(3) In order to stabilize tantalum sols, 100 ml. of corn oil, 6 ml. of Tween 80, 2 ml. of Span 80, 10 gm. of dextrin, 5 gm. of glucose and 30 gm. of oleated Ta_2O_5 of less than 1 μ. size were mixed in a Waring blender or a Virtis homogenizer. Twenty-five milliliters of distilled water were added to form a water in oil emulsion. Seventy-five milliliters of water were added to reverse the emulsion. This emulsion was transferred to a Manton-Gaulin 2-stage homogenizer fitted with a rubber return tube so that it would operate on 200 ml. The emulsion was recirculated at 10,000 pounds per square inch pressure until the temperature reached 80° C., above which temperature Tween 80 emulsions may break. The liver and spleen of 250 gm. rats could easily be opacified with 1 ml. doses of this emulsion.

(4) These experiments were repeated using 10 gm. of hydroxy-lecithin instead of 8 ml. of Tween-Span with similar results (Fig. 2).



Fig. 1. Hepatosplenogram of 250 gm. Wistar rat after two injections of 1 ml. of tantalum pentoxide and dextrin prepared by method (1).

DISCUSSION

Since the specific gravity of Ta_2O_5 is 8.7 and since sols of very small micellae seem to be more toxic, the primary problem is precipitation of the micellae. This prompted us to mix the Ta_2O_5 with an oil of less density than water. If tantalum oleate could be made into a sol it might be useful. While blood serum is a good emulsifying agent, its use introduces the problems of coagulation of proteins, sterility and blood typing.

Roentgen ray absorption is proportional to the 4th power of the atomic number and thorium of atomic number 90 would be ideal if it were not radioactive. Physical chemists have made only *dilute* colloidal sols of gold, atomic number 79 and tantalum atomic number 73. Thomas, Henry and Kaplan[12] injected mice intravenously with 10 per cent thixotropic tantalum pentoxide sols but the mice quickly died. Their death was attributed to embolism but we believe that death was too sudden to be entirely attributed to embolism and may have been due to a respiratory chemo-reflex,[3] for if rats are kept alive by artificial respiration for a few minutes, they may live for a long time. Freshly prepared sols of tantalum are sufficiently soluble to trigger this reflex. The micellae of tantalum pentoxide sols, by internal bonding (oleation) may become insoluble in hydrochloric acid (Werner's theory). This process is common to a number of elements.[10] McClendon and Gershon-

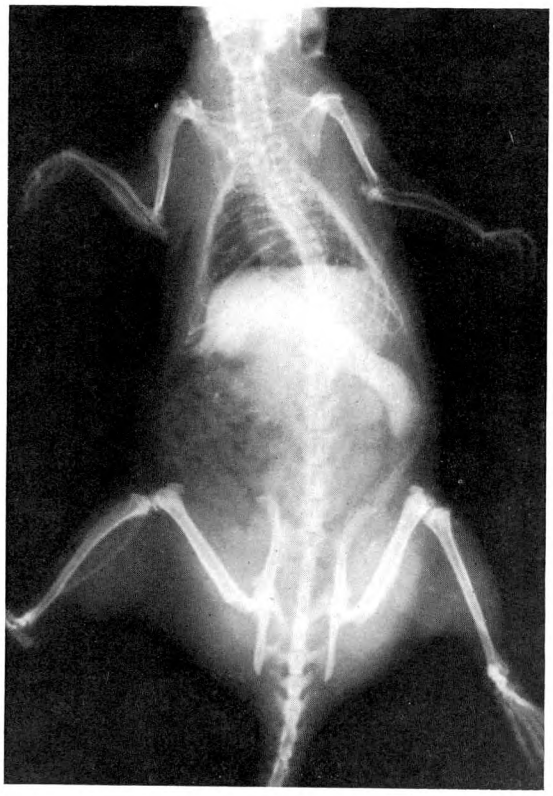


FIG. 2. Hepatosplenogram of rat after single injection of tantalum pentoxide and hydroxy-lecithin and corn oil prepared by method (4).

Cohen[8] observed that oleated tantalum pentoxide of less than 1 μ. size suspended in propyl-oleate emulsion could be injected intravenously into rats in sufficient quantity to opacify the liver and spleen to roentgen rays.

Whether visualization of the liver and spleen is performed by the older method of hepatosplenography or by scintiscanning, loading of reticuloendothelial cells may be desired. Therefore, intravenous injections of insoluble colloids may be useful. Our studies have shown that it is desirable to keep injected volumes low so that any deviation of pH, temperature, osmotic pressure or ionic strength from blood plasma will be greatly lessened by mixing with a larger volume of blood. We have injected 20 ml. of a colloidal preparation that was mainly 5 per cent glucose into a 300 gm. rat with no untoward results. But this has to be done slowly; if the needle slips out before the injection is finished or the anesthesia is too prolonged, the animal may die. All these shortcomings prompted us to turn to radioisotope scanning techniques. At present, we are using a Picker Magnascanner and a 19-hole collimator with 3×3 mm. light shaper for photoscanning.

RADIOISOTOPE SCANNING

SPLEEN

1. Ten milliliters of the patient's blood are withdrawn into a sterile ACD bottle* containing 2 m. of ACD anticoagulant.
2. Two hundred microcuries of Cr^{51} sodium chromate are added.
3. Incubation at room temperature is done for 15 minutes.
4. Fifty milligrams of ascorbic acid are added to reduce residual chromate to chromic ions.
5. The blood is then heated in a water bath at 49.5° C. plus or minus 1° for one hour. After cooling to room temperature the blood is administered intravenously.
6. In 2 hours, the radioisotope scanning of the spleen begins with the patient in a supine position.
7. The patient is marked and a localization roentgenogram is made.

LIVER

Procedure—Rose Bengal.

1. One hundred and fifty microcuries of I^{131} rose bengal is injected intravenously and a scan is performed ½ hour later.
2. Patient is in a supine position. Scan is started at bottom of liver and proceeds cephalad.
3. The patient is marked and a localization roentgenogram is made.

Procedure—Au^{198}.

1. One hundred microcuries of colloidal gold (Au^{198}) is injected intravenously. Scan is done 1½ to 2 hours later over liver.
2. Patient is in a supine position and the scan is performed beginning just above the top of the liver.

* Available from Abbott Laboratories.

3. The patient is marked and a localization roentgenogram is made.

REPORT OF CASES

Case I. N.L., a 46 year old white physician, was admitted to the hospital for the third time. A carcinoma of the colon had been resected approximately 2 years prior to this admission. For several months he had noted fatigue, loss of weight, marked anorexia, sweating, and spooning of his nails. Laboratory examination revealed an elevated sedimentation rate, a normal colon and stomach by roentgen examination and a normal hemogram. Physical examination on admission showed a tender liver enlarged three finger breadths below the costal margin. Liver metastasis was suspected and a biopsy was performed by Vim-Silverman technique and adequate tissue was obtained. The pathologic report, however, was "marked fatty infiltration of the liver." Scanning examination of the liver with Cr^{51} revealed the presence of a large filling defect near the porta hepatis consistent with the presence of intrahepatic metastasis. The patient then underwent an exploratory laparotomy and was found to have carcinoma involving the porta hepatis of the liver (Fig. 3).

Case II. J.V., a 53 year old male, was admitted because of progressive weakness and jaundice of 10 days' duration. A mass was found in the right upper quadrant of the abdomen which was thought to be either an enlarged liver or a pancreatic tumor. A scan of the abdomen with radioactive rose bengal I^{131} revealed a small liver and the mass did not pick up any radioactive material. A presumptive diagnosis of a pancreatic tumor was made. Carcinoma at the head of the pancreas was found at operation and the small liver was found to be due to cirrhosis (Fig. 4).

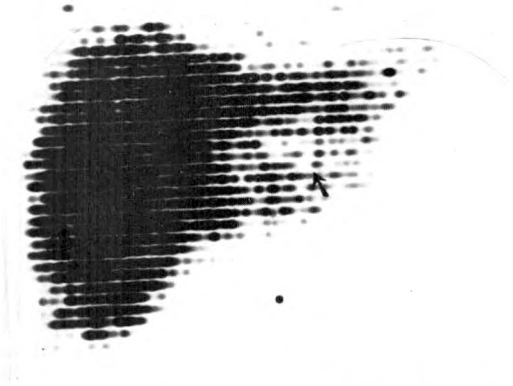


Fig. 3. Case I. Hepatic photoscan revealing metastatic carcinoma.

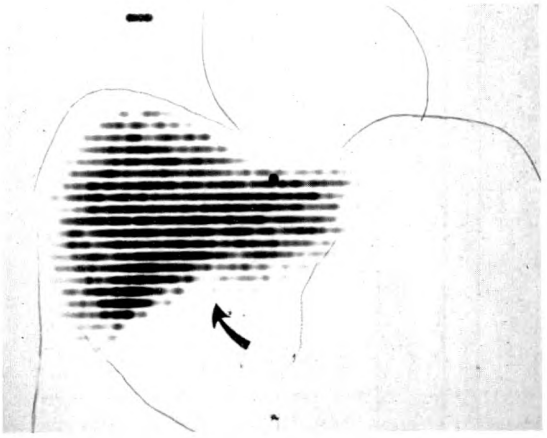


Fig. 4. Case II. Carcinoma of the head of the pancreas accounts for pressure defect on undersurface of liver. Photoscanned with rose bengal I^{131}.

Case III. F.G., a 59 year old white female, was admitted because of persistent pain in the right upper quadrant of the abdomen due to gallstones. A cholecystectomy was performed. In addition to gallstones, a metastatic malignant lesion was found in the right lobe of the liver with extension to the abdominal peritoneum overlying the right lobe. Carcinoma of the colon had been resected in this patient 18 months previously at which time no metastasis was noted in the liver. Calculi in the gallbladder were found during this operation.

Scanning of the liver with radioactive gold revealed a negative defect directly over the area of metastasis (Fig. 5).

Case IV. L.S., a 46 year old white male, had a generalized superficial lymphadenopathy of 2 months' duration. This responded quite well to chlorambucil therapy. Three months later, the patient returned with what appeared to be an enlargement of the liver and spleen. Another course of chemotherapy was carried out without much effect. The patient then was scanned and this revealed the spleen to be normal in size and shape. The large mass in the upper left quadrant which extended 5 cm. below the umbilicus was not an enlarged spleen, but evidently a large retroperitoneal mass corresponding to the original diagnosis of lymphosarcoma made from

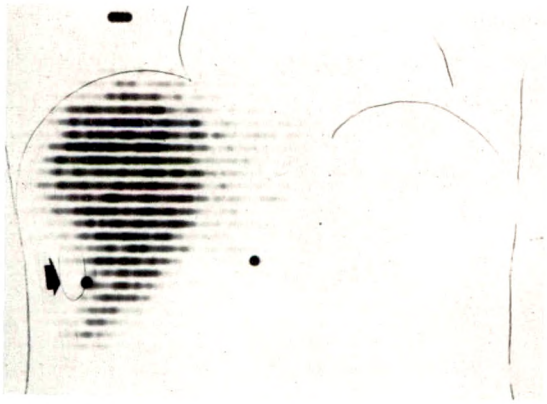


FIG. 5. Case III. Metastatic carcinoma accounts for filling defect revealed by photoscanning of liver with radioactive gold.

biopsy of one of the superficial lymph nodes. Deep roentgen therapy was instituted and the mass in the left abdomen promptly regressed. This occurred one year ago and the patient has remained in good health.

In this instance, the clinicians were not able to differentiate the mass in the left abdomen from an enlarged spleen. Since the scan revealed the spleen to be normal in size and shape, it was obvious that the mass was extrasplenic and it was inferred therefore that the process was probably due to the original disease. An excellent therapeutic result was obtained with roentgen therapy, which is not surprising insofar as lymphosarcoma usually is sensitive to this form of therapy (Fig. 6).

CASE V. D.K., a 60 year old female, was thought to have right-sided cardiac failure with splenomegaly of unknown origin. The scannigram with Cr^{51} confirmed the presence of an enlarged spleen with no appreciable change in its size during a period of therapy lasting one year (Fig. 7).

DISCUSSION

Mechanical stylet and photographic scintillation scanning techniques are hardly more than a decade old,[1,2] but great improvement already has been made in the efficiency of modern apparatus. With the use of TV contrast enhancement, and if laminagraphic techniques can be evolved, scintiscanning will become even more diagnostically valuable in the localization of space-taking lesions in large organs like the liver and spleen.[4,5]

Colloidal gold 198, being concentrated in the reticuloendothelial tissue, is not satisfactory for demonstrating the spleen by scintillation scanning because of the unwanted accompanying image of the liver which traps most of the radioactive colloid. By a method which causes selective splenic uptake of radioactive red blood cells worked out by Johnson, Wood and Mooring,[6] excellent splenic scanning has been accomplished. The splenograms illustrated in this report were obtained by this method, except that the incubation was done at room temperature rather than 49.5° C. Isologous or compatible homologous red blood cells may be used in all subjects regardless of Rh genotype. This method is rapid and reduces the likelihood of contamination of red blood cells and there is rapid uptake by the spleen of the Cr^{51} tagged cells. Photoscanning is preferred by us because it increases appreci-

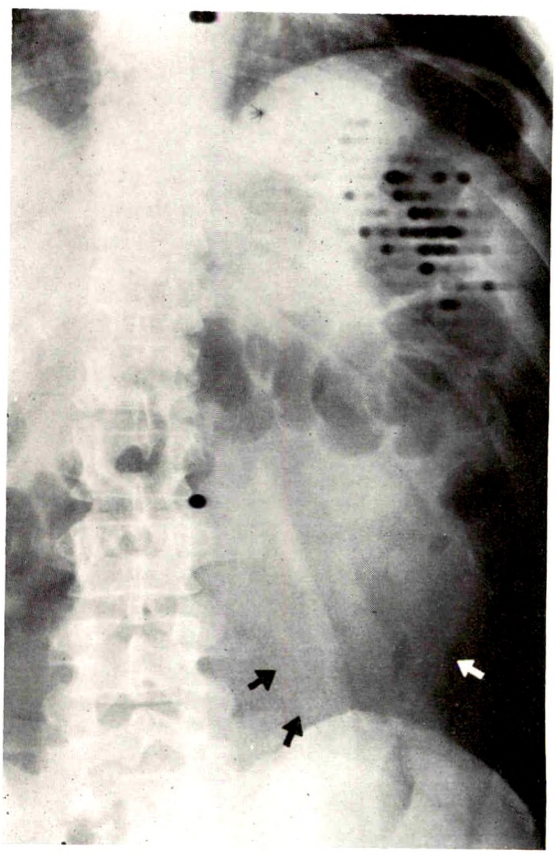


FIG. 6. Case IV. Retroperitoneal mass shown to be outside spleen with which it was confused before Cr^{51} photoscanning of the spleen.

ation of the splenic configuration and permits better superimposition of the roentgenographic image of the spleen. The defined area has sharp margins and its configuration fits quite well the roentgenographic projection of the spleen. Thus the splenic area can be sharply differentiated from any masses in or around this organ.

The hazards of this method are negligible. The danger of inducing significant antibody formation is eliminated by using the patient's own blood. The calculated total radiation dose to various tissues after administration of 200 μc of Cr^{51} bound red blood cells is probably no more than 75 millirads; to the ovary about 80 millirads; to the testes about 25 millirads and to the liver about 400 millirads. The spleen receives probably about 15 rads, due principally to sequestration of most of the radioactive red blood cells.

In photoscanning of the liver, using 100 μc Au^{198} as the maximum dose, approximately 5 rads are delivered to the organ. This is far safer than the use of thorotrast which delivers a dose to the liver estimated to be of the order of 700 to 15,000 rads.[7] We already have found photoscanning with Au^{198} particularly useful in the differential diagnosis of abdominal masses, the differentiation of obstructive parenchymatous liver disease and the detection of malignant lesions of the liver. If the spleen takes up the isotope, depression of the reticuloendothelial system of the liver should be suspected. This might occur with infectious hepatitis or cirrhosis.

The scan qualities obtained with I^{131} rose bengal are not as good as with Au^{198}. Moreover, a loading dose of rose bengal must be employed to avert rapid excretion by the liver.

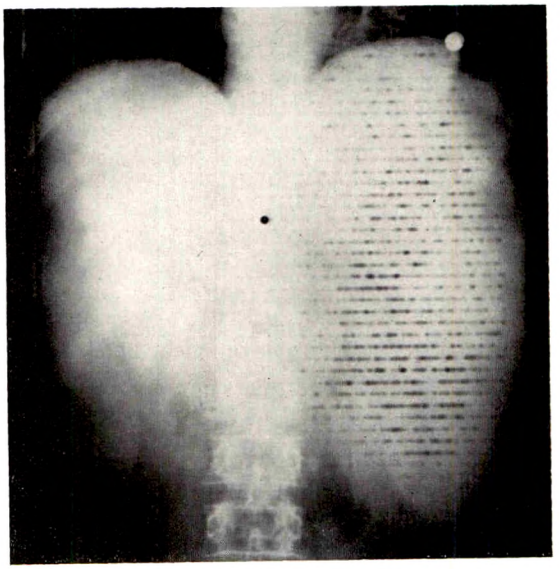


Fig. 7. Case v. Splenic photoscan of enlarged spleen confirming roentgenographic impression.

SUMMARY

During the past decade, we have solved the problem of making nontoxic colloidal sols of tantalum pentoxide and other heavy metals for hepatosplenography in small animals.

The advances made with scintiscanning and photoscanning techniques during the past decade point to fruitful methods of obtaining evidence of disease of the liver and spleen. Most promising is the advent of new and better means of detecting malignancy, not only on the surface, but also in the depths of so large an organ as the liver without resorting to exploratory laparotomy. The spawning of new apparatus, the imaginative experimentation with new types of collimators, the increasing availability of a wider range of more versatile isotopes and the adaptation of television techniques signalize emergence of a bright new diagnostic field in nuclear medicine. Some early achievements in this area are illustrated in this report.

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RADIOISOTOPE RENOGRAPHY*

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RADIOISOTOPE renography, as performed in our laboratory, is a reproducible and sensitive screening test for many types of urinary tract pathology. Through recent changes in the radioactive test material, the instrumentation and the position of the patient, renography can now be used with confidence in the clinical practice of medicine.

Since the introduction of renography in 1956,[10,12] its potential usefulness has been evident. By external body counting, information on individual kidney function and excretion is available quickly and atraumatically. In practice, however, the problem of obtaining accurate, reproducible tracings has been formidable. As late as 1960, Dollery[2] concluded that the test is "unsuitable as a screening test." He suggested that it "is quite possible that the use of other chemical compounds or alteration in technique may surmount some of the difficulties. . . ." Such changes have since been applied to renography, greatly simplifying its use.

The most fundamental change is in the radioactive test material. The first substance applied to radioisotopic kidney function testing was I^{131} labeled iodopyracet (diodrast). In practice it was quickly found that diodrast is in part taken up by the liver; therefore, when detecting probes are placed directly over the kidneys, the right curve is skewed.[2,13] In order to prevent liver uptake of radioactivity, two approaches were taken. One was to block the hepatic uptake of diodrast by adding an intravenous load of nonradioactive diodrast.[1] The desired result was obtained, but simplicity was sacrificed. The second was to use a material which is excreted rapidly by the kidneys only. I^{131} labeled urokon, miokon, renografin and hypaque were not appreciably taken up by the liver but their rate of extraction from the blood was slow[13] thus dulling differences in kidney uptake and excretion.

Finally, a test material was reported which circumvented these difficulties and which has proven in subsequent trials to be an ideal test agent. This is I^{131} labeled iodohippurate (hippuran), which is rapidly removed from the blood by the kidneys and has essentially no hepatic uptake.[4,5,11] The use of hippuran allows the external probes to be aimed directly at the kidneys without elaborate techniques to circumvent or block the liver, while at the same time retaining a high level of sensitivity.

The second change in renography is in instrumentation, and derives from having a test agent which is not taken up by the liver. Early monitoring probes used for obtaining the renogram had small apertures, with the detecting crystal placed close to the skin over the kidneys. Positioning was accomplished by preliminary abdominal roentgenograms. This had the advantage of using less radioactivity but it had the marked disadvantage that small differences in kidney position made considerable differences in the obtained curves.[7,14] Theoretically, the farther the detector from the source of radioactivity, the less difference position changes of the source make. A satisfactory compromise in practice is to withdraw the detecting crystal 6 inches from the skin surface and provide a wide crystal view while maintaining interkidney shielding.[6] The application of this simple principle has greatly improved comparability of kidney function and excretion as well as reproducibility of results.

The third change in the technique for

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renography concerns the position of the patient. Originally, the test was done in the sitting position, mainly because of its convenience. However, when the patient is upright, the right kidney usually drops a varying distance and in some individuals it may be displaced into the pelvis. We therefore place the patient in the supine position, in which the kidneys have little tendency to relocate. Most of the previous geometric problems have thus been eliminated.

METHOD

The scintillation detectors used are a pair of 1 × 1 inch thallium-activated sodium iodide crystals, each feeding through a photomultiplier tube to a separate rate meter-recorder system which describes a curve of the radioactivity changes in its respective area. Each lead collimator measures 2.5×6 inches at the skin surface, sloping back to the crystal which is recessed 6 inches within the collimator. With the patient in the supine position, the centers of the collimators are placed 3 fingerbreadths above the iliac crest, bringing the crystal opposite the usual kidney position (Fig. 1). If an abdominal roentgenogram or intravenous pyelogram is available, the probes are directed over each known kidney site. Responses of the detectors are matched by testing against radioactivity placed at a distance from the probes. When possible,

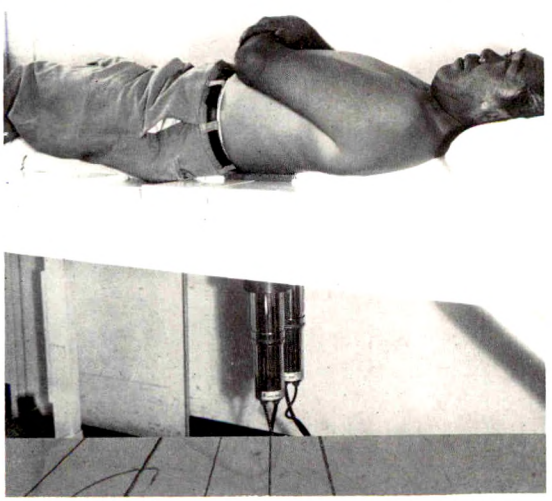


Fig. 1. Position of patient and detecting probes.

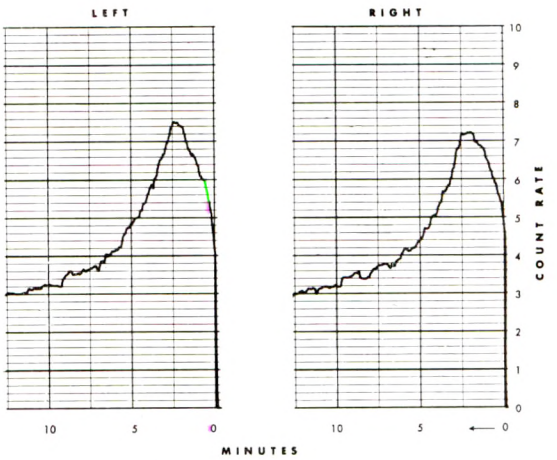


Fig. 2. Normal renogram.

the patient is hydrated with 1 l. of water starting 1 hour before the test. After the intravenous injection of 25 to 40 μc hippuran (depending on the instrument scale as well as the body size and contour), a curve is obtained over each kidney area. If a difference occurs between the two sides, roentgen verification of kidney position is obtained and the test is repeated with probes placed over the known positions.

RESULTS

Normal Renogram. The normal renogram is shown in Figure 2. After the intravenous administration of labeled hippuran, there is a rapid increase in activity beneath the detecting probe, followed by a slower increase which reaches a peak in 2 to 4 minutes and then declines.

The rapid upstroke represents filling of the vascular space. Its height depends on many variables, including the amount of radioactivity given, the blood volume, the distance and substance between kidney and crystal, and the amount of blood in both the kidney and extrarenal areas detected by the crystal. We therefore put little emphasis on its absolute height.

The slower increase phase represents uptake of activity by the kidney and is a result of both tubular function and renal blood flow. Inasmuch as hippuran in trace amounts is extracted from the blood almost completely with each pass through the kidney,[9] the slope of the curve would seem

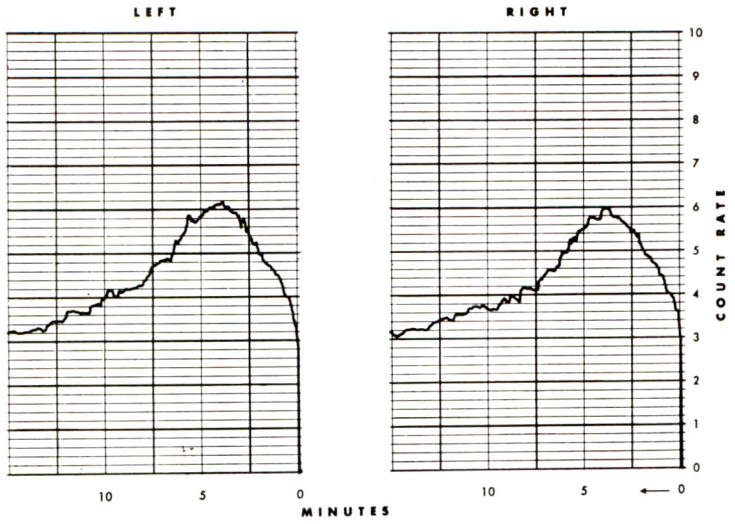


Fig. 3. Renogram of a patient with moderately severe arterial hypertension.

to depend mainly on kidney arterial flow.

The declining curve represents excretion from the kidney area, which includes the kidney and its pelvis.

Bilaterally Decreased Kidney Function. Kidney dysfunction such as is present in moderately severe hypertension or in chronic glomerulonephritis is reflected as a decrease in the second phase; that is, a slower rate of rise after the initial filling. Such a curve is seen in Figure 3, indicating decreased "function" with a correspondingly decreased excretion rate.

Marked kidney dysfunction, such as occurs in severe hypertension or nephritis, is reflected as an almost absent second phase. The curves approach the results expected from a blood disappearance rate (Fig. 4).

Unilaterally Decreased Kidney Function. Figure 5 demonstrates relatively normal function on one side and decreased function on the other.

Unilaterally Absent Kidney. Figure 6 is a renogram from an individual with a surgically absent left kidney. Interkidney shielding almost completely eliminates radioactivity detection from the opposite side.

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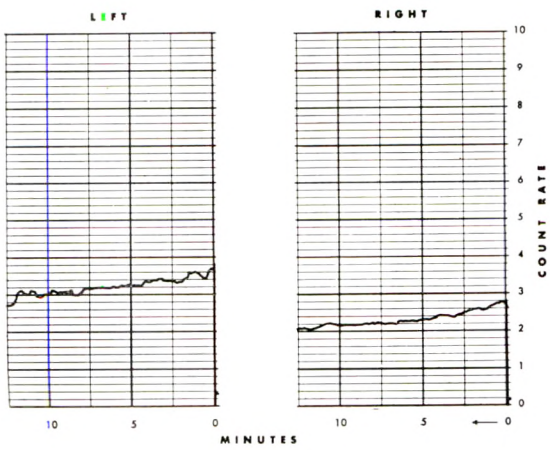


Fig. 4. Renogram of a patient with severe hypertension and uremia.

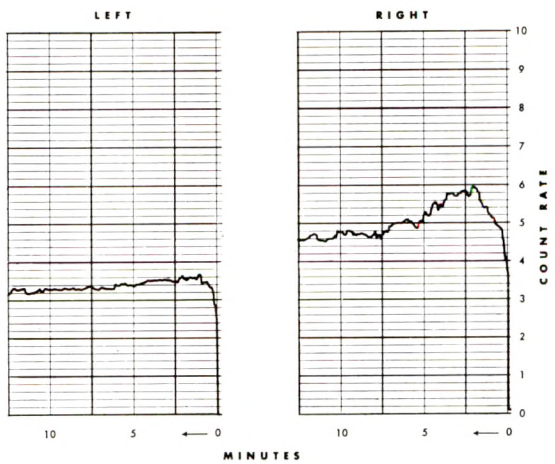


Fig. 5. Renogram of a 21 year old man with recent onset of hypertension. An obstruction of the left renal artery was later found at surgery.

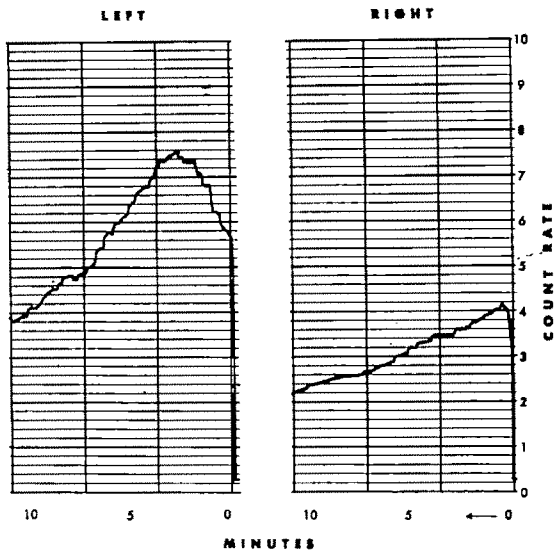


FIG. 6. Renogram of a patient with a surgically absent right kidney.

urinary outflow tract above the bladder level usually results in a rising curve on the obstructed side which is approximately the mirror image of the blood disappearance curve (Fig. 7). Prolonged obstruction, or obstruction which occurs to a kidney with decreased function, may produce a relatively flat curve.

DISCUSSION

With an improved radioactive test agent (I^{131} hippuran), improved instrumentation, and the use of the supine position, confidence in renography has increased greatly. Since it is rapid, atraumatic, sensitive and reproducible, it would appear useful for screening purposes.

Perhaps the most important use of renography is in screening hypertensive individuals for unilateral kidney disease. Accumulating evidence indicates that the renogram is more sensitive than the intravenous roentgenographic urogram as an indicator of decreased function.[8] Compared to the "Howard test," renography is much simpler and less traumatic. Studies in which both tests have been carried out on the same individual suggest that the renogram is as sensitive in determining differences in kidney blood flow as the Howard test;[3,14] verification of this awaits long-term study with improved test procedures. Aortography, in its present state, is not appropriate for a screening test, although it is probably the most definitive procedure for localizing renal ischemia. We thus consider the renogram as a method for determining which hypertensive individuals should be further studied.

Our approach to the patient with hypertension is as follows: First, a renogram is obtained. If this indicates bilaterally equal function and excretion, we proceed no fur-

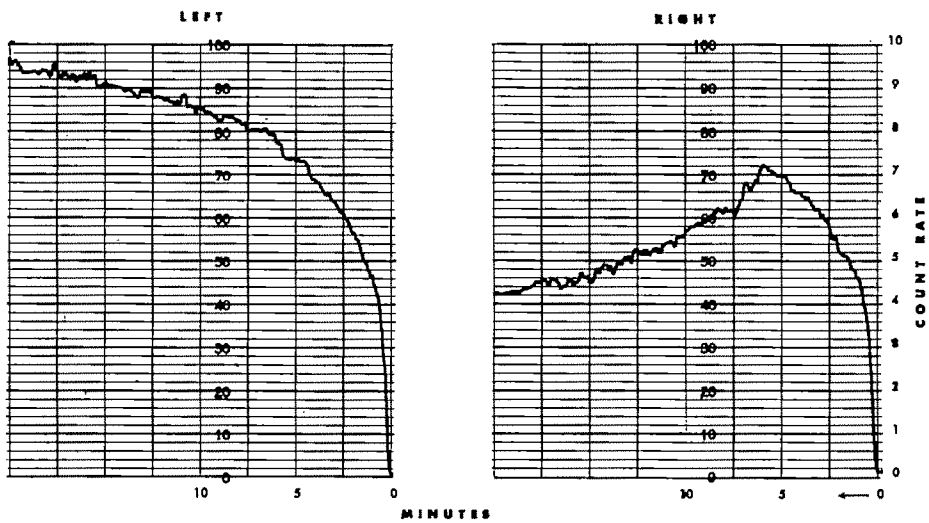


FIG. 7. Renogram of a patient with left ureteral obstruction caused by a stone.

ther unless clinical findings strongly suggest unilateral disease. If there is a decrease in "function" on one side (the slow-rising second phase), we obtain an intravenous urogram and subsequently obtain a repeat renogram with the aid of roentgenographic localization. If either the intravenous urogram or the repeat renogram is abnormal, we proceed to aortography. To date we are allowing aortography to provide the final information, although evidence suggests that both positive and negative errors are made by this procedure.[3]

Renography also provides a rapid screening test for ureteral obstruction. It quickly determines whether a block is present. Once found, it can be followed serially. The attributes of very low total body irradiation (50 μc gives approximately 0.02 rads to the bladder wall and much less to the total body) and rapid blood clearance (65 to 85 per cent is excreted in 30 minutes in the normal individual) suggest its usefulness in pregnant women and children as compared to other irradiation techniques. Its simplicity and sensitivity make it useful in estimating function in each kidney separately before a nephrectomy is done, and in detecting obstruction in severely ill, postoperative and uremic patients.

Renography has become a useful adjunct to standard urologic procedures. The modifications herein described allow its use with increased confidence in clinical practice.

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THE SET-UP FOR A RENOGRAPHY PROGRAM*

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THE ability of an organ or tissue to selectively extract, concentrate and/or excrete a given chemical or drug enables the physician to study that organ. When the drug can be tagged with radioactive atoms, much smaller quantities can be used and often external sampling can be carried out, with minimal disturbance of the patient's equanimity. Winter and others[7,8,9] have shown the way to study kidney function with tagged drugs excreted by the kidneys.

The radio-iodopyracet renogram presents a graphic record of individual kidney function obtained rapidly and without trauma by means of a relatively simple external procedure.[3,5,6,7,8,9] The study is capable of contributing to the diagnosis of renal disorders and of giving considerable functional information otherwise acquired by excretory urography, aortography or bilateral ureteral catheterization.[6,7,9] Complete urologic work-up can be time-consuming, expensive and a painful experience for the patient. Accordingly, a practical service is performed in establishing the presence or absence of renal dysfunction with radio-iodopyracet prior to the institution of the more complicated standard diagnostic procedures. Patients with abnormal renograms are candidates for extensive urologic examinations, while those with normal renograms can be spared the discomfort and expense. The reliability of the radio-iodopyracet renography in detecting disorders of the individual kidney appears from earlier clinical study to be of a high order.[3,5,6,9]

The dynamics of renal uptake and excretion of radio-iodopyracet permit the consecutive assessment of renal vascular capacity, tubular cell function, and the patency of the upper urinary passages.[2,4,6–9] The renogram is divided into 3 distinct segments, which are representative of these fundamental aspects of kidney physiology. After the injection of radioactive material, infiltration of the vascular network of the kidneys and adjacent tissue produces a steep initial spike in the tracing. The height to which these spikes rise is an index of the capacity of each renal inflow system. Secondary rises then occur, the steepness and height of which reflect the quality of tubular cell secretory function. In some instances, recent urinary stasis may modify this segment of the curve. The third and final portion of the curve is normally a drop in the height of the tracing consistent with the ability of the upper urinary tracts to move urine and drug to the bladder. This may be delayed by urinary stasis or by tubular cell dysfunction.

The diagnostic renogram is obtained by an intravenous injection of a tracer dose of radio-iodopyracet (diodrast) and continuous external measurement of the changing radioactivity levels over each kidney area for from a minimum of 10 to a maximum of 30 minutes. Gamma-ray scintillation detectors coupled through rate meters and continuous writing recorders are employed to register the data in the form of permanent tracings. Two sets of radiation detection and recording equipment are necessary, one for each kidney. The results are immediately interpretable. The tracings thus obtained are then plotted on single cycle semilog paper for ease of evaluation. Although this step is not necessary, it provides a permanent record for inclusion in the patient's chart.

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Radio-iodopyracet for this study was supplied by Dr. Paul Numerof, The Squibb Institute for Medical Research, New Brunswick, New Jersey.
† Presently at the Guthrie Clinic, Robert Packer Hospital, Sayre, Pennsylvania.

EQUIPMENT

Figure 1 shows the basic set-up described in this paper. A list of the purchased items follows:

1. Scintillation detectors with straight base collimator 1 inch lead, Nuclear Chicago—Model DS-5-2 1×1 inch sodium iodide crystal.
2. Rate meters, Nuclear Chicago—Model 1620A.
3. Recorders, Texas Instrument—Rectiriters with clutch for variable speed adjustments allowing 0.75, 1.5, 3.0, 6.0, and 12.0 inches per minute or hour chart speed.

The stand was constructed by the University of Virginia Hospital maintenance department. The stand and equipment are portable and can be wheeled throughout the hospital for use in operating rooms, recovery room, and at bedsides. The stand

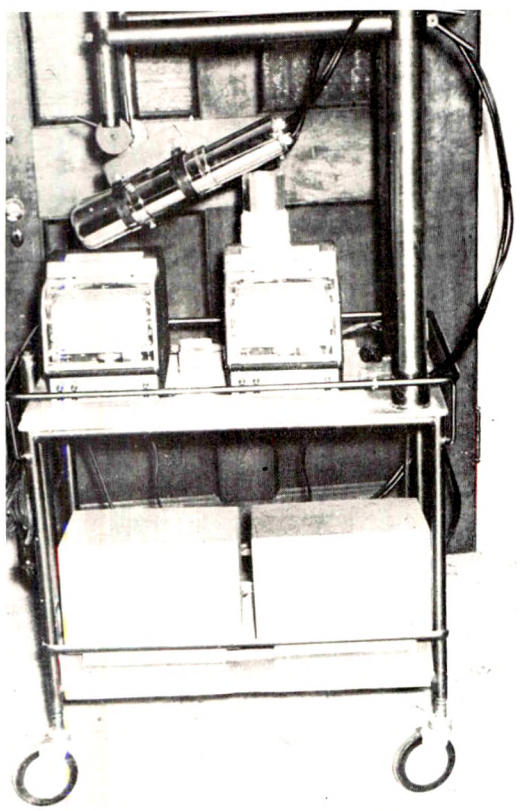


Fig. 1. Photograph showing the basic set-up for renography.

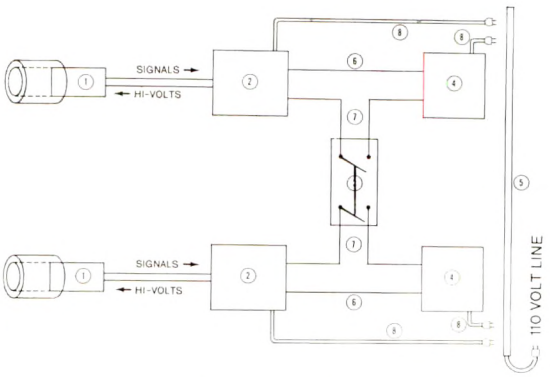


Fig. 2. Electrical circuitry of the system: (1) collimated scintillation crystal; (2) rate meters; (3) double throw switch; (4) Texas recorders; (5) plug mold linear receptacle; (6) connecting wiring (rate meter to recorder); (7) connecting wiring (rate meter through double throw switch to recorder); (8) power line to drive devices.

was designed and used for the study of other dynamic flow and circulatory problems. The health physicist, Mr. K. Williams, devised the electrical circuits so that simultaneous recordings would occur by simply flipping one switch. It is important that both instruments and recorders can be started and run simultaneously. The electrical circuitry is shown in Figure 2.

A few suggestions regarding the selection of equipment are in order. If most of the isotope studies are to be performed with iodine 131, chromium 52, or gold 198, the efficiency of a 1×1 inch sodium iodide crystal is satisfactory. Straight base collimation is good in that the volume covered or seen is well defined and limited. A 2 inch set-back from collimator edge to crystal face further delineates the volume seen. This is shown in Figure 3.

Since this apparatus will be used for other isotope studies, it is advantageous to incorporate as many extras into the system as possible, such as a rate meter with numerous ranges of counts per minute and with many time constants. Although not necessary for the kidney function study, the investigator may wish to incorporate single channel, pulse height counting (or at least base line discrimination) into the set-up. This would enable 2 isotopes to be counted

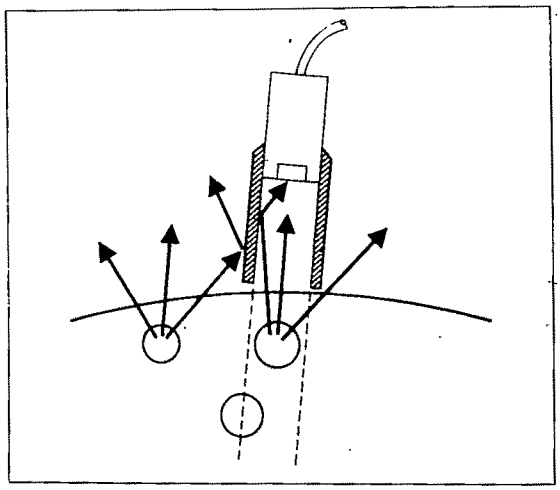


FIG. 3. Diagram showing the scintillation detector with a straight base collimator.

in the presence of each other and eliminate much background interference. A logarithmic rate meter is not necessary but would eliminate some preliminary response testing (as is explained later). The dual rate meter in a single case with the feature of indicating only the difference between the two signals does not appear advantageous for this study and, if the equipment is to be used for other studies, may actually prove to be a limitation. The addition of a third detector rate meter or scaler recorder system to the described system seems to have no particular advantage.

It is important to be able to move the detectors in all planes and degrees. Therefore, if a stand is constructed in the hospital workshops, one should make certain that the following movements are possible (Fig. 4): (1) the position of the detectors in relationship to each other should be from 4 inches (center to center) to 66 inches; (2) the vertical distance traveled from 24 inches to 60 inches from the floor; (3) the movement to and from stand should be 30 inches (need 5 inch cables); and (4) the detector angulation should be 360° in the vertical plane and 240° in the horizontal plane.

PRELIMINARY TESTING AND PREPARATION OF EQUIPMENT

After receiving the equipment, the apparatus should be assembled and physi-

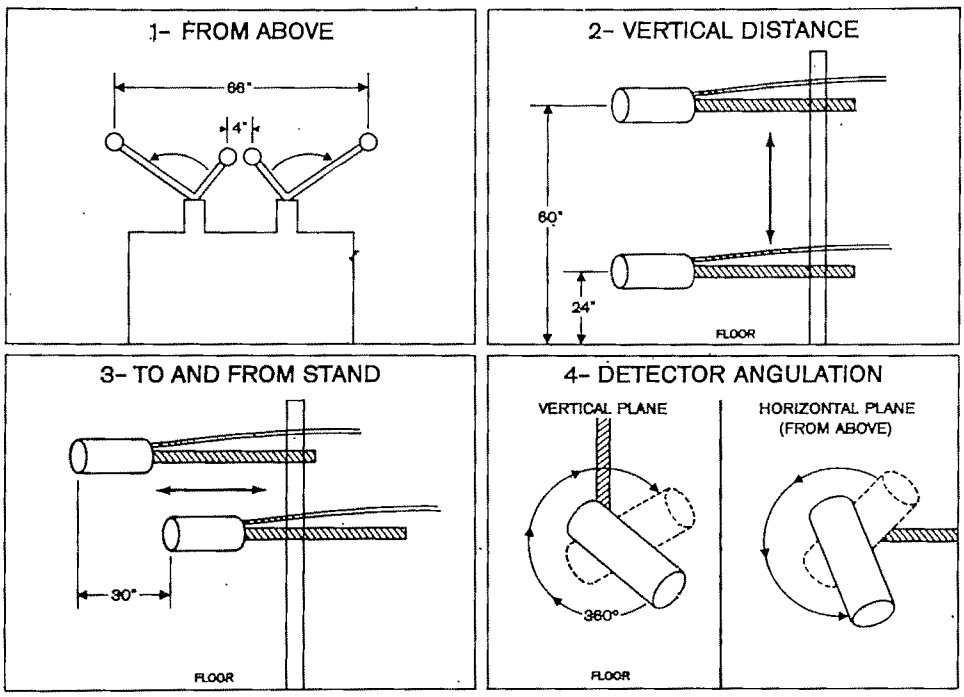


FIG. 4. Diagram showing the recommended construction of the stand.

cally tested to determine the operating voltage (plateau curves) and individual response of each detector, rate meter, recorder, and system. The voltage may not be identical for each detector. The electronic component must be allowed a satisfactory warm-up period to allow stabilization to occur. It is our practice to leave the system "on" but not at operating voltages during the work week. There should be at least a 20 minute warm-up period of the detector at operating voltages.

After the equipment is warmed up and ready to run, it is essential to determine whether the rates of travel of the recording paper and the recording stylus are uniform so that the detectors sense and signal equal responses to the same stimuli. With a radiant source in a fixed geometric relationship to the detector head, the height of the recorded impulse and the rate of ascent should be uniform and identical. It may be necessary to try several detectors in order to obtain a "matched pair." The manufacturer can do the preliminary matching. The signals coming from the detectors to the rate meter must be interpreted uniformly by the rate meters. In turn, the information sent from the rate meter to the recorder must be uniform. The recorders should be checked for lag in one of the styluses. This must be corrected. Basically, the equipment must be adjusted so that a given stimulus results in an identical response on the recording paper for each hemisystem.

CHART SPEED

Chart speed is a matter of personal preference, economy and the desirability of including the tracing with the patient's record. A rate of three inches per minute was usually employed for the first one-third of the study. More rapid rates spread the record out and finer detail can be noted, but may prove confusing. Initially, the completed record ran 60 inches. When we reduced the speed after 5 minutes to $\frac{3}{4}$ of an inch per minute, the tracings were more easily handled and interpreted. The count rate was plotted against time on single cycle semilog paper, in duplicate, for inclusion in the patient chart and isotope laboratory records. This additional step also allows easier comparison between several repeat runs.

EQUALIZATION RESPONSE

In our experience, one of the chief disappointments early in the program was when a recording proved inadequate because a stylus was not working properly. To eliminate this, the detector should be calibrated for various quantities of the isotope. A table can easily be produced which would indicate the following parameters:

Isotope	*Quantity*	*Time Constant*	*Count Rate*	*Other*
Iodine	10 μc	2.0 seconds	30 K	$\frac{c/m}{2}$

These data will enable the nuclear physician to pre-set his equipment before the study or test to be certain of recording all of the information. There are two alternatives: one is to use a logarithmic rate meter in which the signals coming from the detector are compressed logarithmically; the other is to record the signals as interpreted by the rate meter on a magnetic tape for later playback and proper recording.[1] These will increase the cost of the equipment.

DOSAGE

Patients examined at the Isotope Laboratory of the University of Virginia Medical Center were given radio-iodopyracet in a concentration of 1 μc of activity per 3 kg. of body weight (2 to 5 μc/kg. was tried). While this was found to be the optimum dosage for our equipment, allowance had to be made for the slightly higher requirements of the obese individual and for lower dosage in the asthenic patient. The suggested dosage is listed below:

Pounds	*Kilograms*	*Dose in μc*
200	90.9	30.3
175	79.5	26.5
150	68.1	22.7
125	56.8	18.9
100	45.4	15.1

The total administered dose is, therefore, relatively small and well within dose sched-

ules approved for uptake studies. Since the iodine 131 is rapidly eliminated from the body (80–90 per cent within 12 hours), the study is within safe tolerance limits. The accumulated dose of radiation administered with radio-iodopyracet is approximately 1/1,000 that required for a kidney, ureter and bladder study. The 10 to 15 per cent accumulation in the liver is also excreted and poses no problem for the patient. Therefore, the test with radio-iodopyracet can be done on any age group without *undue* concern for radiation dose.

DRUG PREPARATION

The radio-iodopyracet used in our preliminary studies was supplied in 5 or 10 cc. quantities of diodrast tagged with iodine 131 in activity ranges of 90 μc/cc. to 250 μc/cc. Because the dose schedule resulted in the patient needing around 20 μc, the initial stock was diluted on arrival with sterile saline. The prepared working concentration was 100 μc/cc. A tuberculin syringe was used to deliver a dose which ranged from 0.2 cc. initially to 1 cc. When radioactive decay necessitated more than a 1 cc. dose, a 2 cc. syringe was used. When the program was established, a new shipment was ordered every 2 weeks.

When a patient had been subjected to excretory urography, several hours were allowed to elapse prior to the administration of radio-iodopyracet since overloading the renal tubular cells with contrast medium is known to alter the pattern of renal uptake and excretion of the tracer dose, thus invalidating the renogram obtained.

There is dissociation of I^{131} from the diodrast and this free iodine will be picked up by the thyroid gland. It is estimated that a maximum of 10 per cent of the administered dose may find its way to the thyroid. This quantity will pose no physiologic threat. It can be prevented, if Lugol's solution is used beforehand to saturate the free iodine spaces. Lugol saturation was not used in these studies.

Although the quantity of drug injected is 40 to 50 times less than normally used in intravenous pyelography, the nuclear physician should be prepared to meet any emergency resulting from a sensitivity reaction to iodine or the radioiodopyracet.

Syringes, adrenalin, and antihistamine should be on hand. A sensitivity test was not made in our studies; but testing depends on the preference of the physician.

PATIENT POSITIONING

The position of the patient can be sitting or reclining, either on his side or prone. The preferred position is to have the patient sit in a chair and lean over its back as illustrated in Figure 5. This facilitates equipment set-up and the conduct of the study is made easier. It also allows easy access to hand veins for the administration of radio-iodopyracet. The collimating device of the detector should touch the patient's back.

The best tracings are obtained when the detectors "see" only the kidney mass. To achieve this, a preliminary erect roentgenogram of the abdomen is made (kidney, ureter and bladder). The skin area where each kidney is thought to lie is marked with ink and then covered with a suitable radiopaque material and the exposure is made. The relative position of the kidney masses,

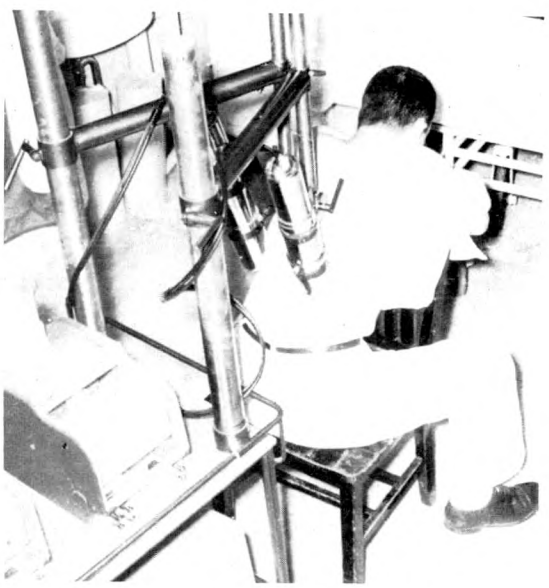


Fig. 5. Photograph showing the preferred position for renography.

the skin marks and the marked roentgenogram is coordinated so that the collimated detectors can be so placed as to "see" only the kidney masses. The angle of the detectors in relationship to the patient's back will vary with the position of the patient. An easy way to judge this angulation is to visualize or project the volume seen by the collimated detector and align this central axis with the skin marking and kidney (Fig. 6). Once alignment has been achieved, the patient must be cautioned not to move. Consideration must be given to hepatic uptake and retention of radioiodopyracet which may interfere with obtaining useful renograms. This necessitates a caudad angulation of the detectors.

A brief résumé of the renographic study as it is performed in the Isotope Laboratory at the University of Virginia follows:

1. The equipment is warmed up at least one-half hour prior to the scheduled study. Tests are made to determine whether the system and all equipment are functioning properly.

2. The patient reports to the Department of Radiology for a single kidney, ureter and bladder roentgenogram. The kidney areas are estimated and marked with ink on the skin in the same position in which the patient is to be studied. These areas are covered with lead markers for protection and the patient and the roentgenogram are sent to the Isotope Laboratory.

3. The patient is positioned and the detectors are placed as indicated (Fig. 7). The relative positions of the kidney, the skin marks and the marked kidney, ureter and bladder are correlated and the detectors are centered over this area. The dose to be used is calculated and the syringe is filled.

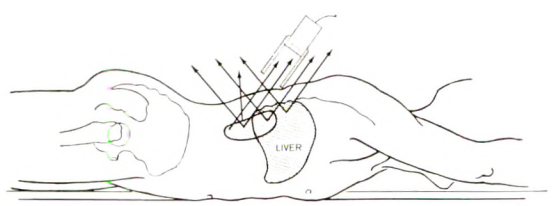


Fig. 6. Diagram showing the placement of the detector so as to obtain a proper renogram of the area.

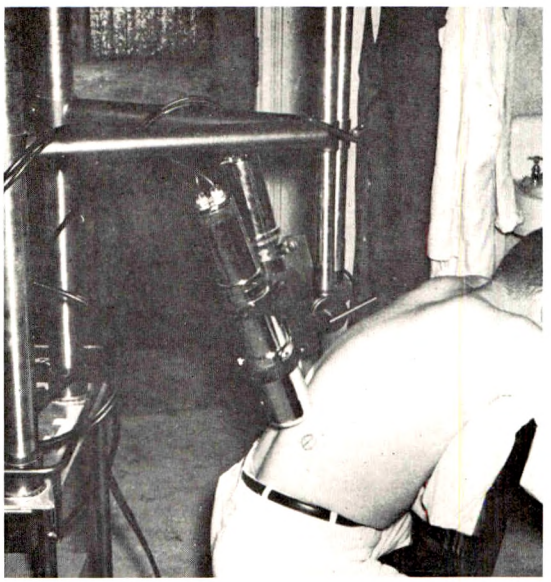


Fig. 7. Photograph showing a close-up of the position of the patient, the skin marks and the detectors.

4. The equipment is started and a 1 minute background is recorded. The filled syringe is placed against the patient's epigastrium until the detectors signal that a peak has been reached. (This step can be eliminated after the study becomes routine, since it is intended only as a final check.)

5. The dose is injected into a dorsal hand vein rapidly; usually a tuberculin syringe and a 21 gauge needle suffice. The point of injection is marked on both tracings.

6. The study is concluded in 15 to 30 minutes, depending upon the progress of the record. After the first 5 minutes, the chart speed can be reduced.

Two factors significantly influence the accuracy and reproducibility of the renogram: (1) it is advisable to calibrate the instruments to respond equally to a standard source of gamma radiation and recalibration with a known standard is recommended prior to each study. The most likely source of error and/or disappointment is in the improper placement of the counters in relation to the kidney areas

(Fig. 7); (2) each detector must be carefully placed so that both will cover equal volumes of tissue, a pre-test kidney, ureter and bladder roentgenogram will help to localize the kidney areas for precise placement of the detectors. Failure to observe these factors leads to difficulty in obtaining a diagnostic renogram.

The set-up of the equipment and some of the physical pitfalls have been indicated. Modifications can be made both in complexity and design to suit the individual nuclear physician. All the leading commercial suppliers of isotope equipment have specially designed renographic units. The prices, including stands, range from $5,000 to $10,000.

CLINICAL EVALUATION AND INTERPRETATION

It is not the intent of this paper to show the value and diagnostic capability of the radio-iodopyracet renogram in clinical medicine. This was initially done by Winter[8,9] and others. Boyd *et al*[2] have shown good correlation between the renogram and other accepted renal studies. When blood chemistry irregularities suggested further diagnostic work up, excretory urography and/or retrograde pyelography generally confirmed the presence of suspected pathology. In the older individual correlation was not as good; abnormalities were found on the renogram which were not found on the intravenous pyelogram. The study is an excellent screening test for kidney disease and has proven itself in accuracy and diagnostic capability. The most difficult portion of the study is the interpretation of the results so that intelligent help can be given the referring clinician.

THE NORMAL RENOGRAM

The normally functioning kidneys yield tracings such as those presented in Figure 8, *A* and *B*. The three-phase curve is characteristic of the normal renogram and modifications are, in sequence, significant indications of abnormal renal vascular

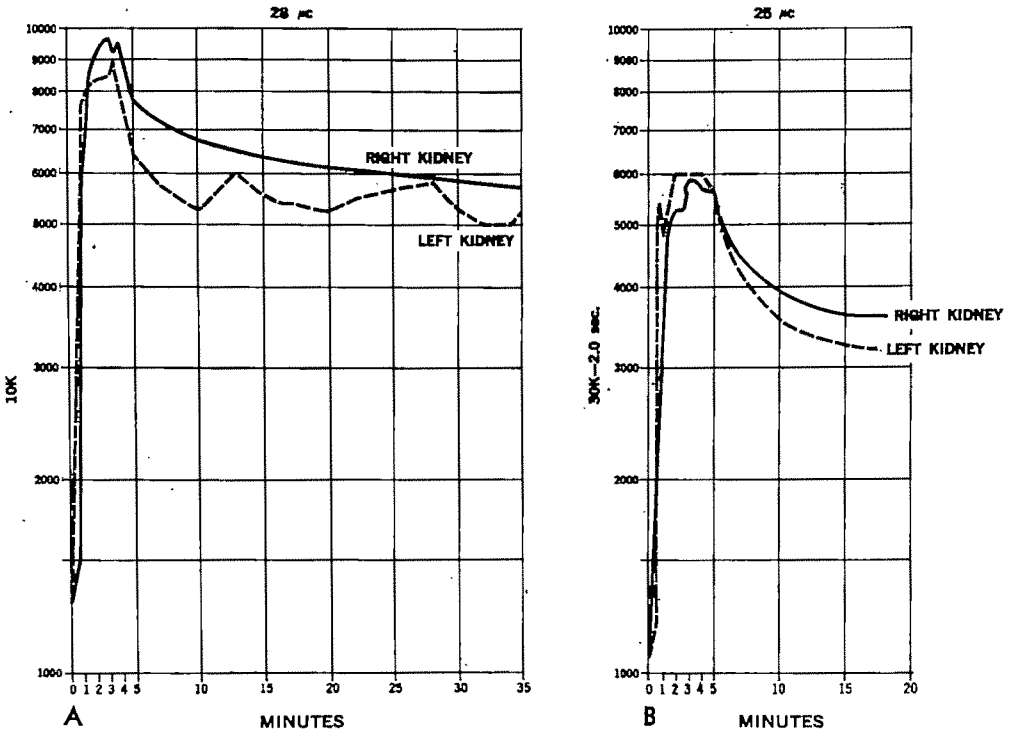


FIG. 8. (*A* and *B*) Tracings on single cycle semilog paper. These are renograms of normally functioning kidneys.

capacity, a defect in tubular cell function, or an obstruction to the outflow of urine in the upper urinary passages. During the study, it was noted that there were slight differences between the slopes of excretion in the segment of the curves for the right and the left kidney. The final portion of the right kidney tracing was less steep usually and reached a plateau at a higher level than the same segment of the left kidney. Still another observation concerns the initial or the vascular segment on the renogram. In younger patients, the initial spike was found to attain a greater height than in older individuals, probably because of the differences in the vascular beds. The remainder of the curve was not visibly influenced by age. A familiarity with the normal renogram gained through experience facilitates its interpretation. The neophyte investigator will have difficulty interpreting his results until he acquires this experience and is able to detect the amount of shading necessary for abnormal curves.

The author found that interpretation was facilitated by transcribing the tracing data onto single cycle semilog paper. Figures 8, *A* and *B* and 9, *A–D* represent this mode of presenting the data.

THE RENOGRAM IN KIDNEY DISEASE

The usefulness of the renogram as a diagnostic test is largely dependent upon accurate interpretation of the deviate curve. The ability to recognize irregularities on the tracing and to associate them with a specific pathology may be gained by obtaining and examining renograms of cases where diagnosis of the renal disorders has been firmly established. Figure 9, *A–D* shows patterns interpreted as abnormal and encountered during the present study. Still others may be found in the reports of other investigators.[2,6,8,9] It is the intent of the author that these curves serve only as guideposts to learning. Urinary obstruction, either partial or complete, is a relatively frequent occurrence. In patients with acute or recent obstruction, the third phase of the curve for the affected kidney rises for a prolonged period of 20 to 30 minutes or longer before reaching a plateau. The excretory down slope is absent (Figure 9, *A* and *B*). With partial ureteral obstruction and hydronephrosis, the height of the initial vascular segment for the affected kidney varies with the size of the renal vascular bed. In cases of long-standing obstruction, the initial spike is abnormally low as a consequence of decreased renal parenchyma blood flow, back pressure of urine, or both factors. The secretory segment is prolonged owing to a decreased rate of removal of urine from the pelvis and impairment of tubular function. The excretory segment of the curve is usually present and the downward slope appears to indicate primarily the degree of ureteral patency.

In nephrosclerosis, renograms consistently show a diminished initial vascular spike, a decrease or absence of the secretory segment, and an abnormal excretory segment (Fig. 9*D*).

The nonfunctioning kidney has available for uptake and secretion of radio-iodopyracet no more than a minimal number of tubular cells. Consequently, only the vascular background is reflected on the renogram of the affected kidney. The secretory and excretory segments are absent (Fig. 9*C*).

Diagnostic features of the curve obtained in acute pyelonephritis include a flattened prolongation of the secretory segment and a decrease in the slope of the excretory segment for the involved kidney. After treatment is undertaken, the tracer test provides a simple means for following the progression of improvement in tubular cell function and renal excretion.

The renogram is, perhaps, of major clinical use as a rapid screening procedure for detecting the presence of renal pathology in the patient with hypertension. By measuring renal vascularity, tubular cell secretory function, and upper urinary tract stasis, the tracing permits analysis of the three functions etiologically significant in renal hypertension. Moreover, comparison of right and left renograms readily identifies the diseased kidney. Evidence of vascu-

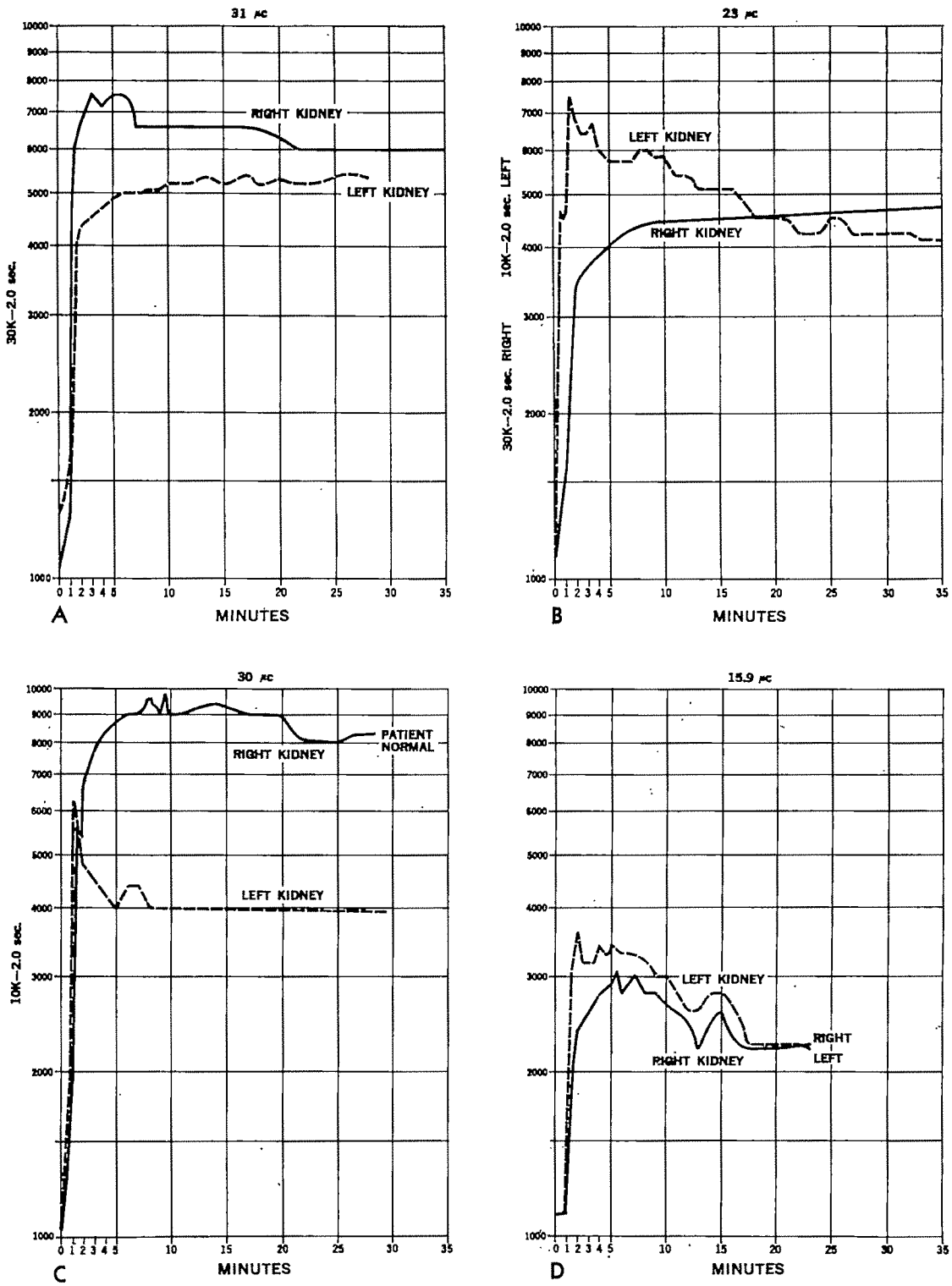


FIG. 9. (*A*, *B*, *C* and *D*) Renograms showing kidney pathology. *A* and *B* show obstruction; *C* a nonfunctioning kidney and *D* nephrosclerosis.

lar deficiency and functional impairment indicates further study to localize and determine the cause of the difficulty. On the other hand, the hypertensive patient showing bilaterally normal renograms may be considered free from renal lesions which might initiate or contribute to blood pressure disturbances.

In previous work with radio-iodopyracet,[6] of 410 patients with normal renograms, 8, or 2.0 per cent, were subsequently found by standard methods to have kidney disease. This high order of reliability was confirmed by individual kidney clearance tests conducted during the investigation.

COMMENT

The radio-iodopyracet renogram has many outstanding features as a renal function test.

1. It is easily and rapidly performed, relatively nontraumatic and without serious complication.

2. Results of the function of each kidney are immediately apparent and recorded permanently.

3. The study does not affect kidney function or renal blood flow; it is a physiologic determination.

4. It can be performed with safety in patients too ill for retrograde pyelography and can be done in children when surgical procedures are not advisable.

5. The radiation exposure dose is approximately 1/1,000th of that from intravenous pyelography and the dose of iodopyracet only a fraction of the quantity used in roentgenography.

6. Good correlation was observed between the findings on the renogram and conventional kidney function tests.

7. Normal bilateral renograms suggested that the patient could be spared the discomfort, loss of time and expense associated with more complicated urologic diagnostic procedures. Until confidence in interpretation develops, the conventional kidney function test should not be discarded.

8. Abnormal renograms provide information as to which phase of renal physiology has been disturbed and needs to be elaborated upon with further investigation. It is an excellent screen for this purpose.

9. The availability of radio-iodohippuric acid will add a new facet to radioactive kidney studies. The use and place of this drug is now being investigated in the Nuclear Medicine Laboratory of the Guthrie Clinic and Robert Packer Hospital. This drug is excreted exclusively by the kidneys and thereby will eliminate radiation from liver uptake.

Although the radio-iodopyracet renogram has exhibited a high order of reliability, the study is subject to 2 important sources of technical error:

1. The positioning of the scintillation counters to avoid inaccuracies introduced by liver uptake and excretion of a small amount of radioactive medium is critical. This may be eliminated by use of radio-iodo-hippuric acid. Proper placement of detecting equipment may be accomplished with the aid of a roentgenogram taken with the patient upright just prior to the radioisotope test.

2. Once the renogram is obtained, it may be erroneously interpreted. A working familiarity with the renogram traced by the normal kidney and by diseased kidneys, affected by the various lesions encountered in clinical practice, is vital. A true translation of the deviate curve into diagnostic information is largely dependent upon experience with the procedure and with the analysis of results. The study of renograms obtained in cases with well-defined kidney pathology is of value to the clinician who wishes to use radio-iodopyracet in the routine screening of patients with suspected renal disorders. It is necessary to perform the procedure initially on numerous normal subjects to establish a reference by which to interpret abnormal patterns traced by various functional or structural abnormalities of the kidneys and ureters.

SUMMARY

The basic equipment necessary to do a

radioactive renal function study has been described as well as the methods and techniques of the physical and clinical aspects of the study.

The radio-iodopyracet renogram is a reliable, rapid, nontraumatic renal function test which provides diagnostic information with regard to renal vascularity, tubular cell function and the patency of the upper urinary passages.

The fundamental usefulness of the radio-iodopyracet tracer test is ascribed to the ease with which the presence or absence of disorders of the individual kidney is revealed, thereby sparing the patient with normal renal function the discomfort, loss of time, and expense of extensive urologic investigation.

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The author wishes to express thanks to Mrs. Howard E. Figg, who provided funds for purchasing the necessary equipment, to Dr. Paul Numerof of The Squibb Institute for Medical Research, New Brunswick, New Jersey for providing the radio-iodopyracet for the initial studies, and to his former associates and colleagues at the University Hospital, Charlottesville, Virginia: Dr. George Cooper, Mr. Kenneth D. Williams and Miss Patricia Barber.

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Vol. 88, No. 2

P^{32} STUDIES OF SPERMATOGENESIS IN MAN*

ESTABLISHMENT OF A SPERMATOGENIC ACTIVITY TEST

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IT HAS been established that mitoses parallel elevated phosphorus need.[10,11,14] It can be assumed therefore that a testicle with one half kilometer long germinal tubules, with about 25 millions of mitoses daily, will concentrate phosphorus. In the sterile testicle with mitotic inactivity, no need for phosphorus exists. It was found that 100 ml. of normal sperm contains 110–120 mg. phosphorus and that the head of a normal spermatozoon contains 3.10^{-9} mg. of DNA in which there is about 9 per cent of phosphorus by weight.[12,13,16]

We decided to introduce P^{32} into the body and to follow its behavior in the testicle by *in vivo* examination of the beta and bremsstrahlung radiation of the superficially situated testis and by a study of the P^{32} excreted in the sperm.

To ascertain the necessary P^{32} dose and the health physics problems involved, we did a trial study on patients with polycythemia treated with P^{32}.[7] It was found that safe, small doses are sufficient for the testicular test. We then carried out the studies on appropriately selected patients.[5]

METHOD

One microcurie of P^{32}, as a sodium phosphate isotonic sterile solution per 5 kg. of body weight, or 10–20 μc for an adult was injected intravenously.† Previous preparation of the patient is unnecessary. One hour later the radiation emitted by each testis was measured separately with a mica window Geiger Müller tube, the glands being brought nearer the surface by stretching the scrotal sac. This result was taken as 100 per cent. We repeated the examination 1, 2, 3, 4, 5, 7, 9, 10 or 15 days later. The radiation dose in subsequent measurements was consistently less. We related it to the one hour result and a curve of testicular P^{32} uptake was plotted. For control purposes the behavior of the P^{32} uptake in another part of the body where multiple mitoses are lacking was examined, *e.g.*, in the nasal region. Thus a second curve, the ethmoidal, was plotted.

MATERIAL AND RESULTS

In 105 patients 132 tests were performed, 238 testicles being examined. Twenty-eight patients were under twenty years of age, 42 from twenty to fifty and 35 over fifty. All patients were checked clinically. In some of them spermatograms, hormonal excretions, the chromosomal sex pattern and biopsies were studied.[9,18,22] The data were compiled and the conclusion was reached that an analysis of the P^{32} uptake curve enables the determination of the presence or absence of spermatogenic activity in the *in vivo* examined testicle. We have, therefore, named the procedure the "spermatogenic activity test" or SAT.

Four types of testicular uptake curves could be distinguished:

1. In testes with active spermatogenesis, there was an uptake of P^{32}, owing to the dividing cells. This resulted in the appearance of a plateau or peak on the third to fifth day. The SAT is positive (Fig. 1).

2. The phosphorus is not taken up by testes in which no spermatogenic activity is present as is not taken up by any other organ without mitoses. No plateau appears. The SAT is negative (Fig. 2).

3. In children under six to seven years of age, in some people of old age (after the age

† For the performance of the first trials we suggest a higher dose of P^{32} (20–30 μc) until experience is acquired.

* From the Radium and Isotopes Institute, Tel-Hashomer Government Hospital, Tel-Hashomer, Israel.
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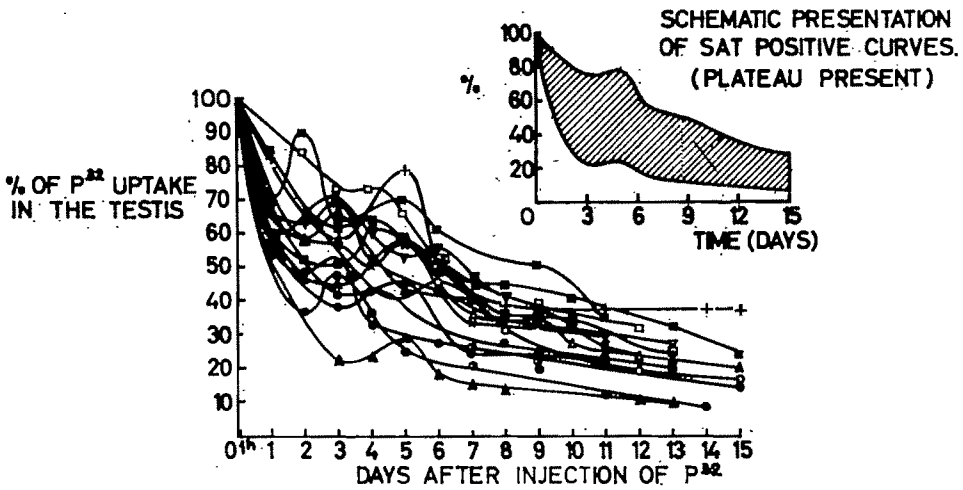


FIG. 1. Curves of P^{32} uptake in the testes of men with active spermatogenesis. Positive spermatogenic activity test (SAT).

of seventy), and in some cases of oligospermia, impotency or hypogonadism we found a retarded and low plateau on the eighth to twelfth day. This is an infantile SAT (Fig. 3).

4. In about 10 per cent of the cases the form of the curve does not allow interpretation. This is an equivocal SAT. A re-examination is recommended.

The correlation between the testicular and the control ethmoidal curve shows that usually, and if the nasal region is normal, the curves intersect in the SAT positive cases and parallel each other in the negative ones (Fig. 4).

Twenty-five patients with various clinical diagnoses tested with P^{32} underwent testicular biopsy. In all but 3 cases the results concurred with the SAT (Fig. 5 and 6). The 3 discrepancies were in 2 cases with oligospermia and 1 case with necrospermia. They were clinically sterile men, their SAT was negative but the biopsy normal (Fig. 7).

Table 1 summarizes the results of the SAT in the 105 patients. The men are classified according to the clinical diagnosis. Figures 5, 6 and 7 show the clinical study of some of them.

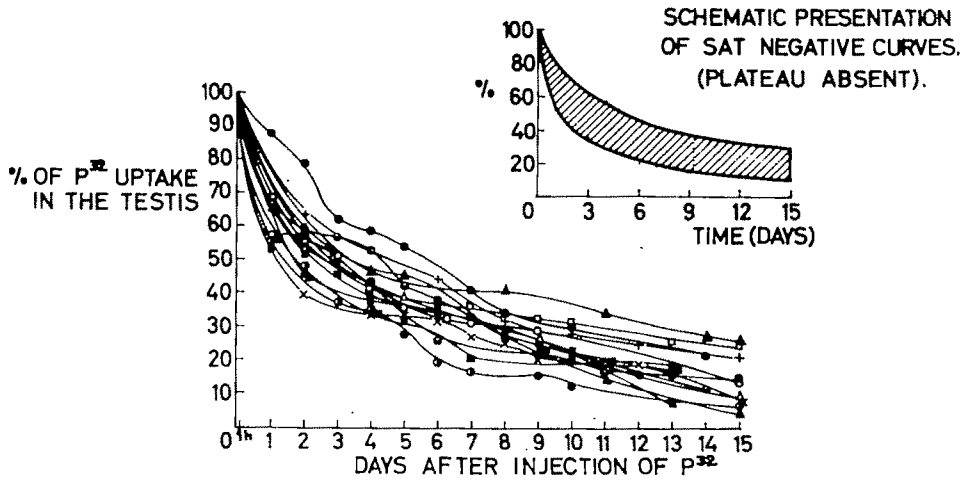


FIG. 2. Curves of P^{32} uptake in the testes of men without spermatogenesis. Negative SAT.

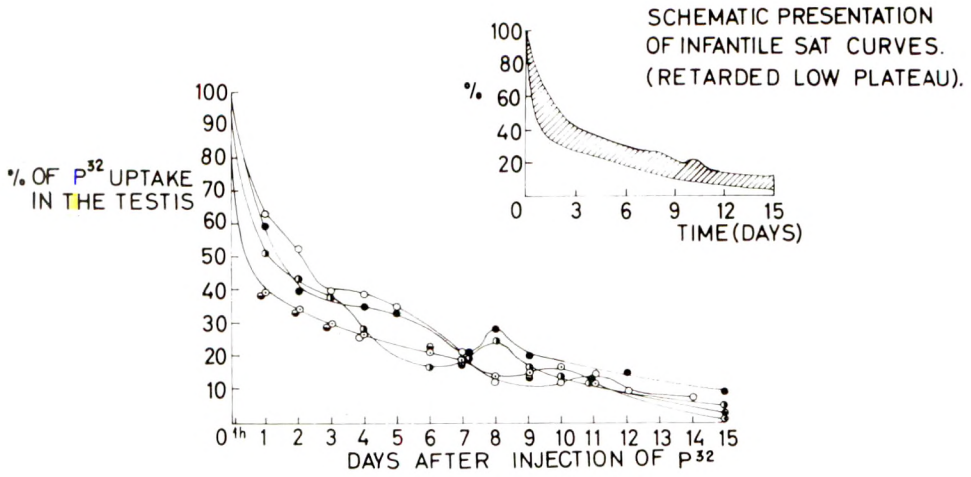


FIG. 3. Curves of P^{32} uptake in the testes with abnormal "infantile" spermatogenesis. Infantile SAT.

RESEARCH PROBLEMS

For more advanced study and research purposes we performed the following additional procedures:

1. Testicular phantoms were constructed. A 1/2,000 part of the injected P^{32} dose was introduced into the phantom and the radiation was measured simultaneously *in vivo* and in the phantom. The percentage of P^{32} uptake by each testis and the biologic half life of the P^{32} could then readily be established. Table II and III give part of the results. The higher P^{32} uptake was noted one hour after the injection and it was found to be 0.05 per cent of the injected dose in the fertile testicle and 0.04 per cent in the sterile and infantile one. Small traces of P^{32} were found up to 110 days after P^{32} treatment. The biologic half life depends on the spermatogenic activity and on the patient's age. It is from 3 to 10 days and more. The more active the spermatogenesis the longer the biologic half life. The younger the patient the shorter the biologic half life (Table III).

2. The ejaculum was collected at different intervals after the P^{32} injection, diluted with Krebs-Ringer buffer solution,[2] and centrifuged. The supernatant, the sediment and a standard sample were incinerated,

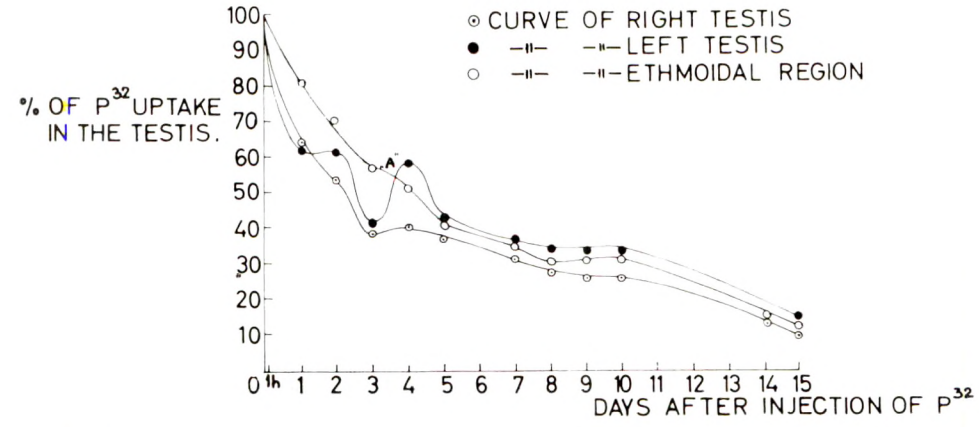


FIG. 4. Correlation between testicular and ethmoidal curves of P^{32} uptake in positive and negative SATs. Patient with an ectopic right and a normal left testis. Right testis: negative SAT; no intersection with the ethmoidal curve. Left testis: positive SAT; the curves intersect at point A.

TABLE I

THE RESULTS OF THE SAT IN 105 PATIENTS

Clinical Diagnosis	No. of Cases Examined	SAT			
		Positive	Negative	Infantile	Equivocal
Normal fertility	28	27	—	1	—
Impotent (with normal testes)	8	4	2	1	1
Aspermia	3	1	1	—	1
Azoospermia	8	—	8	—	—
Oligospermia	10	3	4	1	2
Necrospermia	3	—	3	—	—
Cryptorchidism	13	4	3	3	3
General dystrophy	9	—	—	8	1
Hypogonadism					
Klinefelters	6	—	6	—	—
Testicular atrophy	8	—	8	—	—
Miscellaneous	5	—	3	1	1
Panhypopituitarism	4	—	3	—	1
Total	105	39	41	15	10

TABLE II

PER CENT OF P^{32} UPTAKE BY TESTES AS REVEALED BY SAT (average)

SAT	No. of Testes Examined	Per Cent of Injected P^{32}		
		after 1 hour	after 4 days	after 8 days
Positive	76	0.046	0.031	0.028
Negative	85	0.039	0.020	0.018
Infantile	21	0.041	0.021	0.020
Equivocal	23	0.030	0.018	0.018

TABLE III

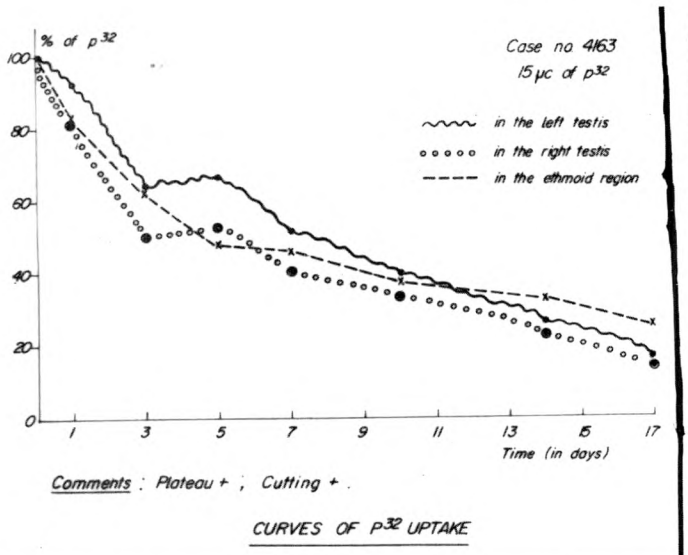
BIOLOGIC HALF LIFE OF P^{32} IN THE TESTES AS REVEALED BY SAT

Spermatogenic Activity	No. of Testes Examined	No. of Cases with Biologic half life (in days)		
		up to 5	up to 10	over 10
Positive	76	20	24	32
Negative	85	45	27	13
Infantile	21	17	4	0
Equivocal	23	11	8	4

TABLE IV

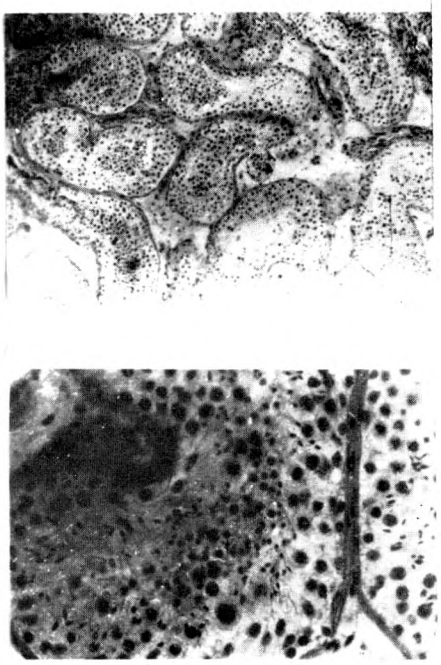
EXAMPLES SHOWING THE RADIATION DOSE RECEIVED BY THE TESTES IN SAT

Spermatogenic Activity	Injected Dose (in μc) (I°)	Per Cent Uptake Testes (F)	Effective Half Life (days) (T)	Mass of Testis (grams) (m)	Dose of Beta Exposure (mrem)
Positive	15	0.05	7.3	20	140
Positive	10	0.05	3.5	15	60
Negative	15	0.04	3.5	10	107
Negative	10	0.04	7.3	15	74
Infantile	3	0.04	3.5	7	31



Uptake of P^{32} in % of the injected dose (in case no 4165)

The examined organ	Time after injection						
	1 h.	1 d.	3 d.	5 d.	7 d.	10 d.	20 d.
Rt. testis	0.056	0.050	0.034	0.042	0.034	0.032	0.027
Lt. testis	0.056	0.057	0.049	0.054	0.043	0.038	0.032
Ethmoid region	0.065	0.058	0.056	0.046	0.041	0.048	0.050
Thigh	0.029	0.013	0.014	0.012	0.010	0.012	—
Glans penis	0.047	0.034	0.032	0.028	0.027	0.026	0.022



HISTOLOGICAL EXAMINATION OF TESTICULAR BIOPSY (case no 4163)

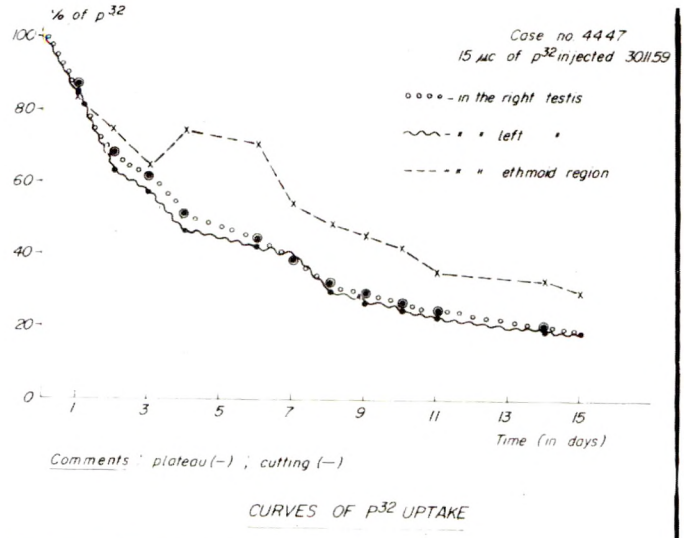
ALL THE SPERMATOGENESIS CELLS AND NORMAL SPERMATOZOA PRESENT

FIG. 5. Positive SAT data and the microscopic findings in a fertile man. Case No. 4163. Sixty-five year old worker, married and father of 2 children. Clinical state: fertile, spermatic ducts patent, operated for hypertrophy of the prostate. Spermiocytogram: $100–120 \times 10^6$ spermatozoa per ml., with 80 per cent motility and 18 per cent pathologic forms.

calcium and phosphorus salts were added and the P^{32} was coprecipitated, filtered, and measured[6] and its quantity calculated. Our preliminary results showed that in 1 ml. of semen there was nearly 0.001 per cent of injected P^{32} on the first and ninth day and 0.007 per cent on the fifth to sixth day in the SAT positive cases. In the SAT negative cases the amount was from 3 to 10 times smaller. In SAT positive cases we found more P^{32} in the sediment than in the supernatant on the fifth to sixth day. On the first day the respective results were inversed.

3. A small quantity of the semen-sediment was covered with a stripping film for a period from one to four months in order to obtain an autoradiogram.[4] This can be done only in patients who had received a therapeutic dose of P^{32} and in animal experiments. Figure 8 is an autoradiogram of semen ejaculated by a patient with polycythemia who five days previously received 3 mc of P^{32}. It shows clearly that at this time the P^{32} is concentrated mainly in the caudal part of the spermatozoon's head and in the intermediate portion.

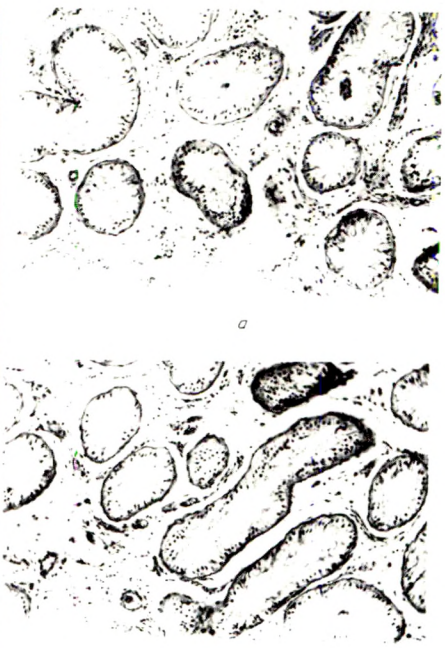
4. An anticipated serious problem was the possible radiation hazard arising from the P^{32}. The total disintegration of 1 μc of phosphorus/gram of tissue corresponds to 600–800 rem of radiation.[1] We calculated therefore that the maximum exposure during SAT is in the order of 100–150 mrem



CURVES OF P^{32} UPTAKE

Uptake of p^{32} in % of the injected dose (in case no 4447)

The examined organ	Time after injection						
	1h	1d	2d	3d	4d	9d	11d
R testicle	0033	0032	0028	0026	0023	0019	0018
L testicle	0034	0032	0025	0023	0020	0017	0016
Ethmoid region	0021	0019	0018	0016	0020	0017	0015



a

b

HISTOLOGICAL EXAMINATION OF TESTICULAR BIOPSY

a = right testis b = left testis

In the tubuli only Sertoli-cells present; the lumen is wide and empty; the basalis is thick and without hyalinization

FIG. 6. Negative SAT data and the microscopic findings in a sterile man who seven years previously was operated on for a bilateral testicular ectopy. Case No. 4447. Twenty-five year old installer, married 4 yr., childless. Clinical state: sterile, presently testes of normal size. Laboratory data: estrogens >40 <100 m. U/l., FSH >33 <50 m. U/l., 17-ketosteroids 9.4–8.3 mg./24 hr. Spermiocytogram: azoospermia.

when 15 μc is injected. We may also utilize the health physics formula:

$$D\beta_{rem} = \frac{74 \cdot F \cdot I^{\circ} \cdot T \cdot \Sigma \bar{E}(RBE) N^{3,16}}{m}$$

All the factors being known, we calculated that the gonadal exposure (average) is 120 mrem (Table IV gives some pertinent evaluations). This approaches the maximum permissible dose for the professional worker/week, and is close to the background radiation received by the general population during one year. Calculations of the bone marrow exposure show a range of 0.1–0.45 rem, the background exposure being 0.15–0.50 rem per year.

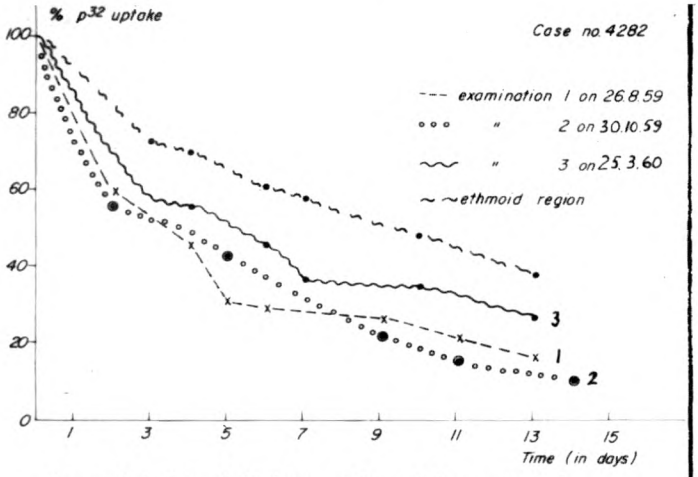
COMMENTS AND CONCLUSIONS

The advantages of the spermatogenic activity test (SAT) are:

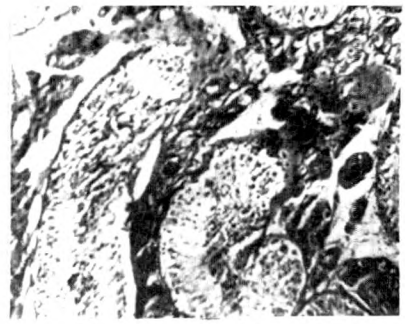
1. The examination can be performed on an outpatient basis, requires no surgery and no semen examination, which is impossible in children, impotents, psychics and others. Each testicle can be examined separately. For clinical diagnosis the determination of the testicular P^{32} uptake alone can be sufficient.

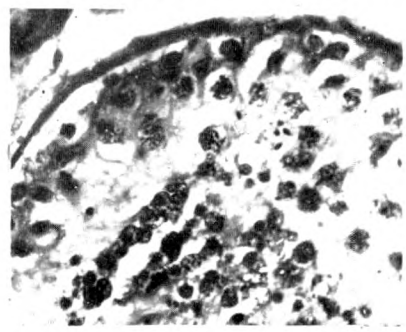
2. SAT can be easily repeated and may be of help in controlling the results of medical treatment and of external or internal influences on spermatogenesis.

3. The SAT can be useful for physio-



CURVES OF P^{32} UPTAKE





HISTOLOGICAL EXAMINATION
In most tubuli:
normal spermatogenesis
In some tubuli:
Sertoli-cells and spermiogenesis absent.
Hyperplasia of the Leidig cells.

Uptake of P^{32} in % of the injected dose on 26.8.59 (case no. 4282)

The examined organ	Time after injection									
	1 h.		2 d.		4 d.		6 d.		11 d.	
	befor treat.	after treat.	befor treat.	after treat.	befor treat.	after treat.	befor treat.	after treat.	befor treat.	after treat.
Rt. testis	0.03	0.05	0.02	0.03	0.02	0.02	0.01	0.014	0.01	0.010
Lt. testis	0.03	0.05	0.02	0.03	0.02	0.03	0.01	0.02	0.01	0.010
Ethmoid region	0.02	0.05	0.02	0.03	0.02	0.03	0.01	0.03	0.01	0.02
Thigh	0.01	0.02	0.005	0.010	0.005	0.008	—	0.007	—	—
Glans penis	0.02	—	0.01	—	0.01	—	0.01	—	0.01	—

FIG. 7. Discrepancy in the findings of a man who was highly oligospermatic but clinically sterile. Three SATs (one before and two after hormonal treatment) were negative; the histologic examination was normal. Case No. 4282. Thirty-two year old teacher, married and father of 1 child, but childless for the last six years. Clinical state: sterile, undescended right testis, atrophy of left testis. Laboratory data: FSH 83 m. U/l., 17-ketosteroids 19.8 mg./24 hr. Spermiocytogram: $0-6\times10^6$ spermatozoa per ml. with 20 per cent motility and 46 per cent pathologic forms.

pathologic studies. The uptake curves and the autoradiograms of the semen have shown, for example, that the duration of spermatogenesis in man is from four to five days.

4. The SAT can be useful in the study of animal reproduction.

5. The test is a physiologic one correlated with the activity of the germinal tubules alone.

The disadvantages of the SAT are:

1. There is technical difficulty since the measurement of the P^{32} uptake must be repeated four to six times and at least for six to ten days.

2. There is a gonadal exposure to ionizing radiation which equals the yearly background radiation.[3] This means in effect that a man tested by SAT can theoretically be considered as being one year older from the point of view of gonadal exposure. It is unlikely that a difference in age of one year in man changes the mutation rate in his offspring.[19] The accepted doubling dose of 50 rem is 500–1,000 times higher than that provided by SAT.[8]

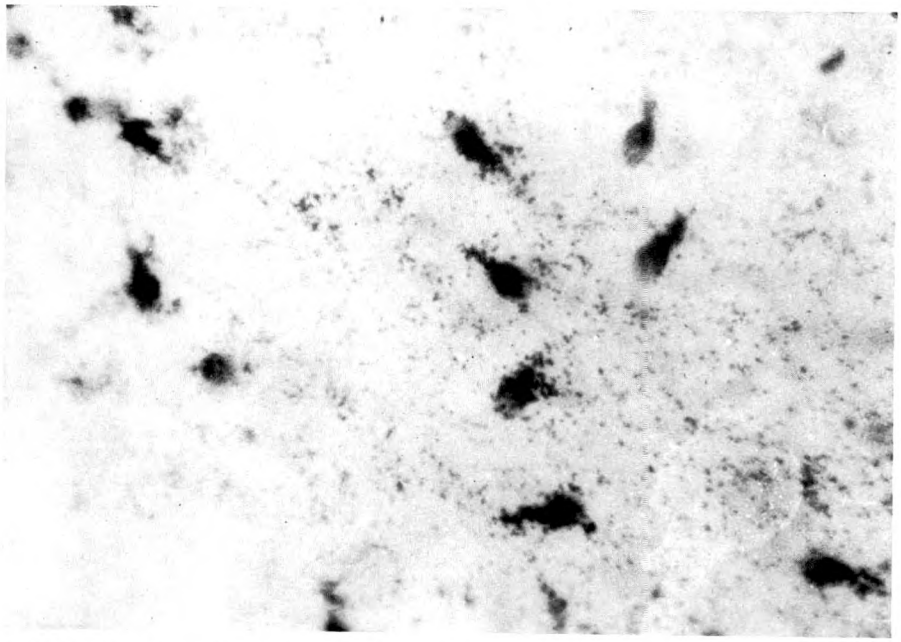


FIG. 8. Autoradiogram of human spermatozoa. A smear of semen of a fertile policythemic man, ejaculated five days after an injection of 3 mc of P^{32}, was covered with a nuclear emulsion.

It may be added that the gonadal dose from roentgen examinations of the pelvis and neighboring parts is higher than that from the SAT (Table v).[12,20,21]

Indications and contraindications must obviously be established. Even considering the low radiation exposure, we emphasize that the SAT is contraindicated in all cases where no need for it exists. The SAT should not be a routine universal examination.

The indications are as follows: (a) patients suffering from functional disturbances of the sex organs, as from sterility, in whom the usual examinations reveal no cause; (b) cases in which standard tests are not possible or refused (children, mentally disturbed persons, neurotics, impotents, etc.); (c) in medico-legal cases where an objective physiologic test of spermatogenic activity is necessary; (d) for assessment of therapy where repeated biopsy is not possible; (e) where it is desirable to test the functional state of each testicle separately; and (f) where research work on the physiopathology of the spermatogenic activity in

TABLE V

RADIATION DOSES TO THE MALE GONADS IN VARIOUS ROENTGEN PROCEDURES

Type of Procedure	Doses in mrem
Femoral arteriography	300–1,360
Cystography	1,500–1,990
Hip roentgenography	460–1,640
Roentgenography of pelvis (anteroposterior)	97–1,140
Pyelography	97– 905
Barium enema study	58–1,420
Roentgenography of lumbar spine (anteroposterior and lateral)	14–2,010
Background gonadal exposure per year	120– 170
SAT per examination	31– 140

special human cases and in different animals requires its use.

SUMMARY

An *in vivo* test for spermatogenesis using P^{32} has been described. In 105 patients the spermatogenic activity test (SAT) was performed and the results were correlated with available clinical and histologic data. The advantages and disadvantages, the indications and contraindications of SAT have been considered.

Special attention has been paid to the possible hazards of the test and they have been commented upon.

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ENCEPHALOPATHIC HYPERTHYROIDISM MANIFESTING AS DYSPHAGIA

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HYPERTHYROIDISM is a protean disease entity with many unusual facets. In their treatise dealing with bizarre clinical manifestations of hyperthyroidism, Chapman and Maloof[1] presented cases with findings of abdominal pain, peripheral edema, heart failure, peri-arthritis of the shoulders, severe myopathy or even the encephalopathic form of hyperthyroidism. Many have attempted to categorize the neurologic manifestations of hyperthyroidism, and the following is an over-all general classification of the different types: (1) chronic thyrotoxic myopathy; (2) hyperthyroidism associated with periodic paralysis; (3) hyperthyroidism associated with myasthenia gravis; (4) exophthalmic ophthalmoplegia; and (5) encephalopathic hyperthyroidism.

(*1*) *Chronic Thyrotoxic Myopathy.* In this group of patients muscular wasting and weakness develop insidiously during the course of thyrotoxicosis. The thenar and interossei muscles are especially affected. Paresis of the larynx and the pharynx is sometimes found, and fibrillary twitchings are the rule; however, there are never signs of pyramidal involvement. There can be intermittent dysphagia with paresis of the muscles of deglutition.

(*2*) *Hyperthyroidism Associated with Periodic Paralysis.* There have been over 40 cases of hyperthyroidism associated with periodic paralysis recorded since Rosenfeld's description in 1902. It has been reported that immediately preceding and during an attack of paralysis, there is an increase in the rate of aldosterone excretion. Primary aldosteronism itself is characterized by spells of paralysis, polyuria, polydipsia, and moderate hypertension. The intermittent aldosteronism associated with hyperthyroidism appears concomitantly with a lowering of the serum potassium level. In addition, the urinary excretion of aldosterone can be markedly elevated with an attack of paralysis.

(*3*) *Hyperthyroidism Associated with Myasthenia Gravis.* Thorner in 1939 first called attention to the observation that when hyperthyroidism and myasthenia gravis are present simultaneously, the hyperthyroidism appears to ameliorate the myasthenia and radioactive iodine therapy aggravates the myasthenia. The true myasthenia of hyperthyroidism does not respond to neostigmine but will respond to therapy for the hyperthyroidism.

(*4*) *Exophthalmic Ophthalmoplegia.* In these cases it has been postulated that the exophthalmos is caused by the thyrotropic hormone. Prostigmine and iodine therapy give no improvement in exophthalmos. Some patients with goiter do not develop ocular proptosis or periorbital swelling and edema, but do have the signs of sympathicotonia, which include lid lag, globe lag, diminished blinking and retraction of the upper and sometimes the lower eyelid.

(*5*) *Encephalopathic Hyperthyroidism.* Waldenstrom[7] states that in acute thyrotoxic myopathy death usually occurs within a week or two of the onset of the bulbar symptoms. The bulbar palsy is associated with muscular weakness of the limbs. The roentgenographic findings cannot be differentiated from those seen in bulbar thrombosis, myasthenia gravis, etc., but they are quite unlike those seen in the Plummer-Vinson syndrome where closure of the larynx and of the nasal cavity is present.

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The decidedly cranial localization of the paresis with impairment of deglutition, sometimes also paresis of the larynx, more rarely of the muscles innervated by the sixth, the seventh or the twelfth cranial nerves is of help in the diagnosis of bulbar paralysis. Before the demise of the patient, there is often coma or symptoms of psychosis. As opposed to these fulminating cases of acute thyrotoxic myopathy, there have been cases with central nervous system involvement which have responded to therapeutic measures employed for hyperthyroidism with a very satisfactory outcome.

REPORT OF A CASE

A 70 year old male was admitted to the thoracic surgery service on February 23, 1959 with a tentative diagnosis of carcinoma of the esophagus, which was based on a history of a fifteen pound weight loss, dysphagia and weakness, developing in three months. The symptoms were progressive. Roentgenographic findings elsewhere tended to substantiate the diagnosis, since barium was seen in the trachea following a barium swallow, although on fluoroscopy a definite fistula was not observed.

Bronchoscopy failed to demonstrate a tumor, and the patient was referred to the Department of Radiology for further examination. A diagnosis of pseudobulbar paralysis was made, since there was definite impairment of the swallowing act and aspiration of barium at the time of fluoroscopy was noted (Fig. 1 and 2). Cinefluorographic studies verified these findings.

Further questioning of the patient revealed that the dysphagia caused coughing, gagging and, at times, aspiration of food or fluid.

Physical examination was unremarkable except for slight tachycardia (108 per minute.) The neurologic findings showed deviation of the protruded tongue to the right, with fasciculations; also, mild sensory and muscular deficit of the fifth cranial nerve on the left. The shoulder girdle muscles exhibited marked atrophy. Fasciculations were noted in the left triceps muscles. Atrophy was seen in the muscles of the hands. All deep tendon reflexes were accentuated on the left, with a positive Hoffmann sign and positive extensor responses.

Four days later, the patient was discharged with a diagnosis of "late amyotrophic lateral sclerosis, associated with mild bulbar palsy, probably the residual of cerebrovascular accident."



Fig. 1. Roentgenogram of the esophagus showing the vallecular sign, with barium remaining in the vallecula, pyriform sinuses and esophagus after a barium swallow.

It was not until August 3, 1959 that the patient was again seen. At this time, he was referred by the Department of Internal Medicine to the Isotope Division of the Department of Radiology for an I^{131} uptake study for probable encephalopathic hyperthyroidism. He had shown further weight loss, dysphagia and extreme weakness, to the extent that he was unable to walk more than a few steps at a time. The I^{131} uptake in twenty four hours was 73 per cent, and this was verified by a seventy-two

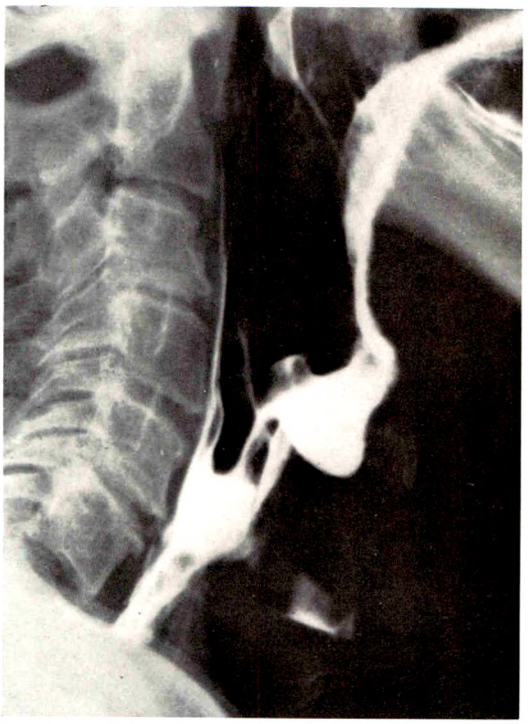


FIG. 2. Lateral roentgenogram showing barium in the pharynx, nasopharynx, vallecula and trachea because of the inability to swallow properly.

hour PBI^{131}. The PBI^{131} equalled 0.35 per cent microcuries per liter. (Normal is less than 0.25 per cent microcuries per liter.) A pulse rate of 120 per minute was noted. The thyroid gland was estimated to weigh about 40 gm. The findings verified the clinician's diagnosis of hyperthyroidism. On August 6, the patient was given an oral dose of 4 mc of I^{131}. He showed rapid improvement, with complete loss of dysphagia, weakness and neurologic symptoms.

On a return visit in October, 1959, the patient had a weight gain of 16 pounds and an I^{131} uptake of 40 per cent. He has been followed at six month intervals and continues to remain in good health with no physical complaints.

SUMMARY

The case presented demonstrates several of the bizarre features of encephalopathic hyperthyroidism. Dysphagia, with pseudobulbar paralysis, weakness, muscular wasting and reflex disturbances may all be symptoms of masked hyperthyroidism. The typical findings of hyperthyroidism in such cases may be absent or of minor concern.

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THE USE OF "TUMOR" AND "BRAIN" PHANTOMS IN THE INTERPRETATION OF POSITRON ANNIHILATION AND UNBALANCE SCANS OF BRAIN TUMORS*

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VARIOUS methods for localizing brain tumors by external gamma ray counting have been used. In recent years a brain scanner was developed by Sweet, Brownell and co-workers[1–5] which makes use of both coincidence counting and unbalance counting. To help in the interpretation of the brain scans obtained by the use of this type of scanner, series of scans were made on phantoms, simulating a tumor in the brain. The volume, position and concentration of the tumor phantom were varied while the volume and concentration in the brain phantom were kept fixed. The results and their application to brain scanning are presented in this paper.

A positron-emitting radioisotope must be used. As^{74} and Cu^{64} were found to be the most appropriate.[1,3] As^{74} was used for our study. A dose of 1.5 mc in the form of sodium arsenate for a 70 kg. person is injected into an antecubital vein. One hour later the head is scanned.

The patient's head is placed on a head rest and centered between the plastic grids marked in a coordinate system. The scanners are then brought up to the plastic grids so that each just clears the plastic as it moves back and forth scanning the patient's head. The scintillation crystals of the scanners now are equidistant from the midplane of the patient's head (Fig. 1).

As^{74} has a half-life of 17 days. It emits beta, gamma and positron radiations. When a positron is emitted, it is very short-lived and travels for a millimeter or less in tissue before it gathers an electron. This electron-positron combination is annihilated and goes off as two photons of .51 mev. energy, 180° apart. The scintillation crystals are connected to a recording circuit so that only when both crystals receive radiation at the same time will they record. This is known as coincidence counting and will occur mostly for positron annihilation radiation. The scintillation crystals are also connected to another circuit for recording gamma radiation. A print is made when one crystal receives a preset amount more of radiation than the opposite crystal. This produces the unbalance scan.[2] These two types of scans, the coincidence scan or positron scan and the unbalance scan, are made simultaneously. The positron annihilation radiation will serve to locate the emitting substance in the plane of scanning, and the gamma radiation in the unbalance circuit will indicate whether the emitting substance is to the right or the left of the midplane.

Figure 2*A* is a positron annihilation scan of a patient's head in a lateral plane, showing where the tumor is located. Each positron print represents 10 coincidence counts. There is a dark area below the brain where the musculature of the face picks up more As^{74} than the brain. Figure 2*B* is the unbalance scan. If the left scintillation crystal receives more radiation than the right, a bar is printed, and if the right receives more than the left, a curved line is printed. Each print represents a difference of 1,500 counts between the right and the left.

These scans are also called positro-

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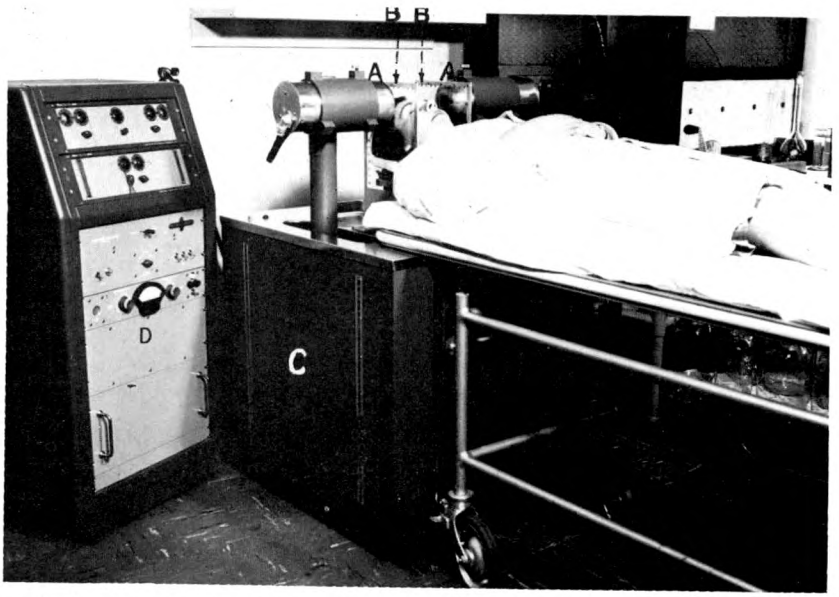


FIG. 1. Positron brain scanner with patient. A-A—Scintillation detectors inside lead shield; B-B—plastic grids; C—printer housing; D—scaler console.

cephalograms and asymmetrogammagrams respectively.

The scans in Figure 2, *A* and *B* are easy to interpret. The positron scan is clear-cut, as is the unbalance scan. However, the unbalance scan does not indicate how far to the left of center the tumor is located. Additional information may be obtained by turning the patient's head 90 degrees and scanning. The positron scan of this antero-posterior view is shown in Figure 3*A*, and the unbalance scan in Figure 3*B*. The lesion is clearly indicated in the positron scan. In Fig. 3*B* the curved prints indicate where there is more activity in the anterior portion of the head, and the straight prints where there is more in the posterior portion.

Most of the scans are not so easy to interpret as these. Frequently, the unbalance scan shows a difference, while the positron

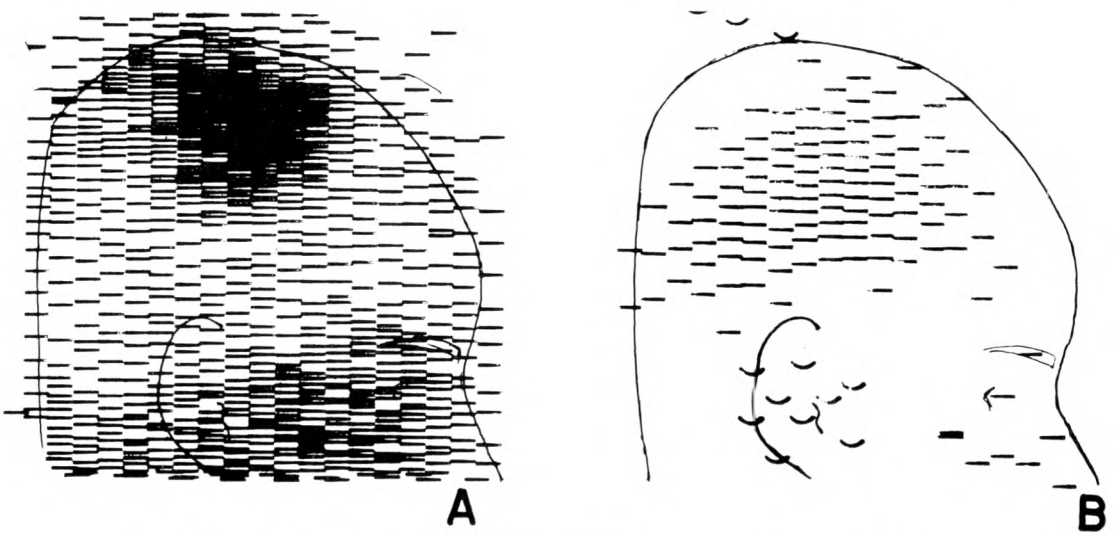


FIG. 2. Lateral view of a patient's head. (*A*) Positron scan. (*B*) Unbalance scan.

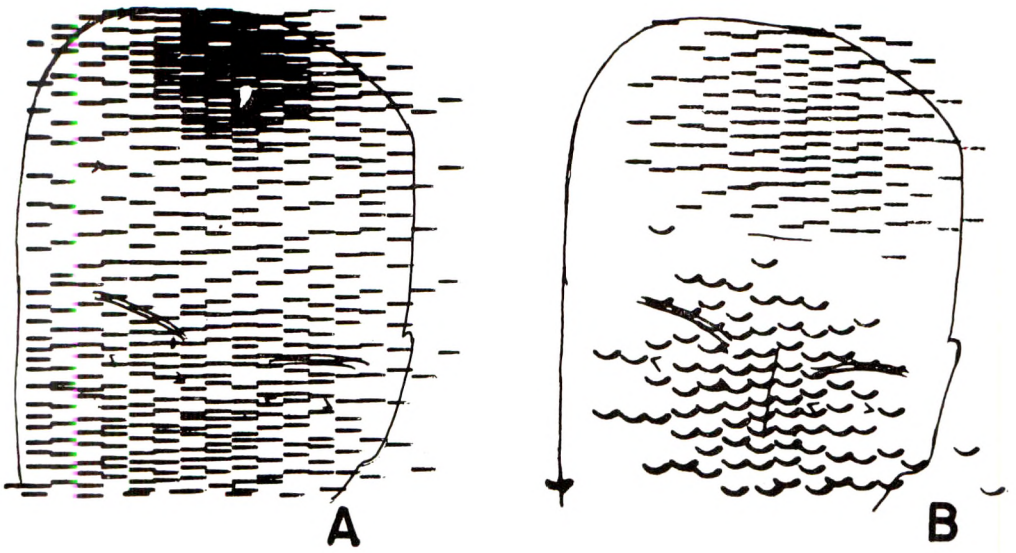


Fig. 3. Anteroposterior view of patient in Figure 2. (*A*) Positron scan. (*B*) Unbalance scan.

scan does not. Sometimes the scan may show a barely discernible abnormality in one position and not in another. Since no work about which we know has been done on phantoms, this investigation was undertaken to find what differences would be recorded by the positron scanner and by the unbalance scanner.

METHOD

A 15.5 cm. diameter battery jar called the "brain" phantom served as a simulated calvarium, and bottles of various volumes suspended from a portion of a meter stick served as a tumor. These "tumor" phantoms could be displaced to the right or the left of the midplane and anteriorly or posteriorly. The experimental set-up is shown in Figure 4. Different concentrations of As^{74} were put into the bottles while the concentration in the "brain" phantom was kept fixed. This concentration was that which was found by scanning to give the same configuration of positron prints as would a scan of a patient's head; it was 6 to 8×10^{-3} μc/cc. (Fig. 5). A positron annihilation scan and an unbalance scan were then made simultaneously for each condition.

The scanner was first balanced with a special device, which we call a spacer, in the center of which was a test tube containing As^{74}, equidistant from the scintillation crystals. Balancing consisted of changing the voltage of the circuits so that the right and the left printer did not print; *i.e.*, the right and the left scintillation crystals received the same number of photons.

RESULTS

1. Effect of Volume and Position of the "Tumor" Phantom on Scans

After preliminary scans were run it was found that a so-called small "tumor" volume of 8 cc. with a diameter of 2 cm.

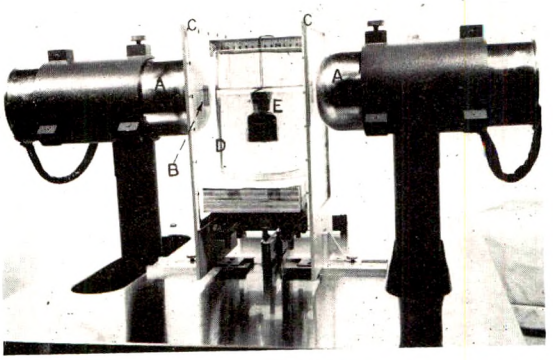


Fig. 4. Experimental set-up of "tumor" and "brain" phantoms. A-A—Scintillation detectors; B—collimator opening; C-C—plastic grids; D—head phantom; E—tumor phantom.

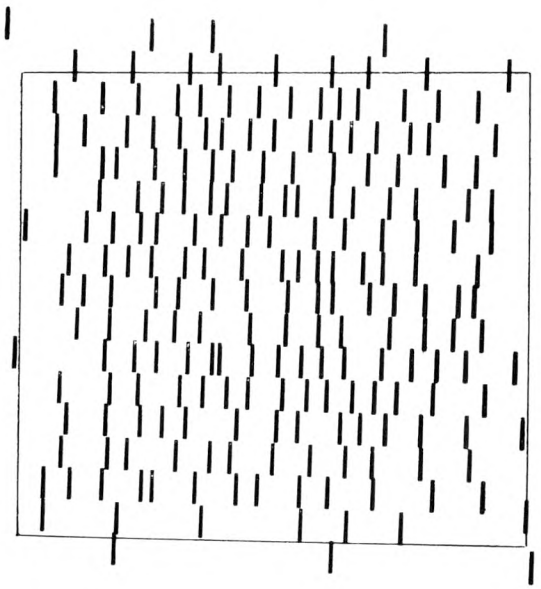


FIG. 5. Positron scan of "brain" phantom with 6.8×10^{-3} μc/cc. concentration of As^{74}.

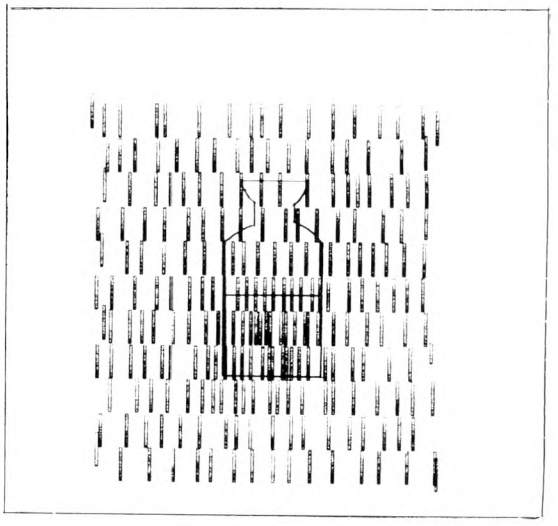


FIG. 7. Positron scan of a 14 cc. "tumor" volume in the center of the midplane with a 15/1 "tumor" to "brain" concentration, 1,100 volts.

needed a ratio of the "tumor" to "brain" concentrations of 15/1 to give a just discernible positron scan of the tumor phantom (Fig. 6). The unbalance scan showed the position of the "tumor" phantom when displaced 2 cm. to the right or left of the midplane, and, of course, for greater displacements. Figure 7 shows the positron scan of a 14 cc. "tumor" volume with a diameter of 2.8 cm. when placed at the center of the head phantom. The positron scan had the same definition when the bottle was moved 5 cm. to the right or the left of the center as when the bottle was at the center. Figure 8 shows the positron scan of a 75 cc. "tumor" phantom.

Figure 9, *A* and *B* shows the unbalance

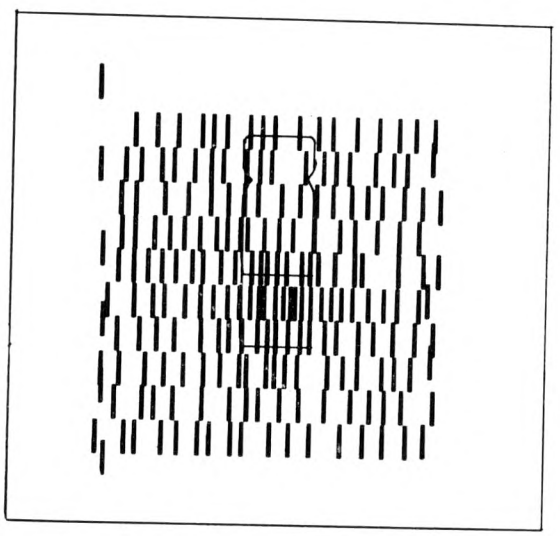


FIG. 6. Just discernible positron scan of 8 cc. volume, 15/1 "tumor" to "brain" concentration, 1,100 volts.

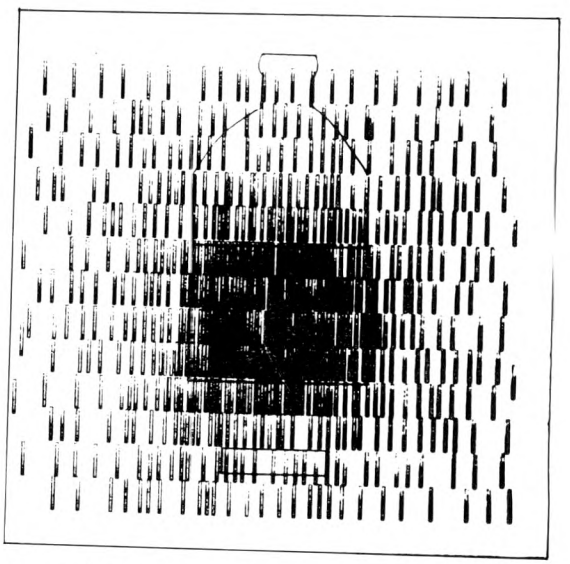


FIG. 8. Positron scan of 75 cc. "tumor" volume, 15/1 "tumor" to "brain" concentration, 1,100 volts.

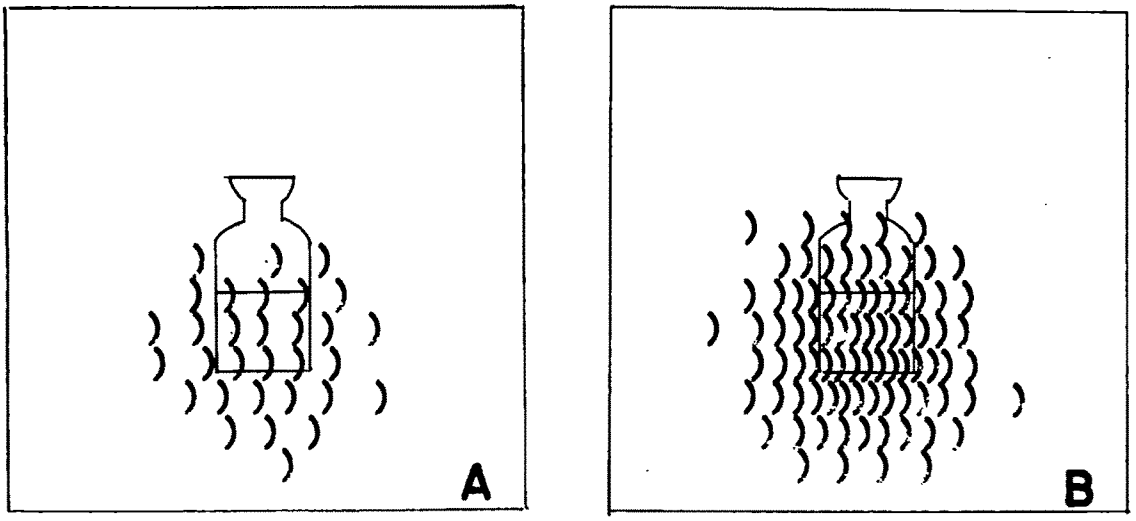


Fig. 9. Unbalance scans of 14 cc. "tumor" volume, 15/1 "tumor" to "brain" concentration. (*A*) 2 cm. right of midplane; (*B*) 4 cm. right of midplane, 1,100 volts.

scans when the 14 cc. "tumor" phantom was moved 2 cm. and 4 cm. to the right respectively. Figure 10, *A* and *B* shows the unbalance scans when the "tumor" phantom was moved 2 cm. and 4 cm. to the left of the midplane respectively. Notice how the number of prints and the area of prints increase as the distance from the center increases. There is poor definition, yet the emitter is approximately in the center of the printed area.

A "tumor" volume of 4 cc. and a diameter of 1.6 cm. with a "tumor" to "brain" concentration ratio of 15/1 was not discernible on the positron scan but could be detected on the unbalance scans at about 2 cm. to the left or the right of the midplane. The unbalance scanner is more sensitive than the positron scanner, but the localization of the tumor is more diffuse.

To find what changes occurred in the scans if the tumor was located in the ante-

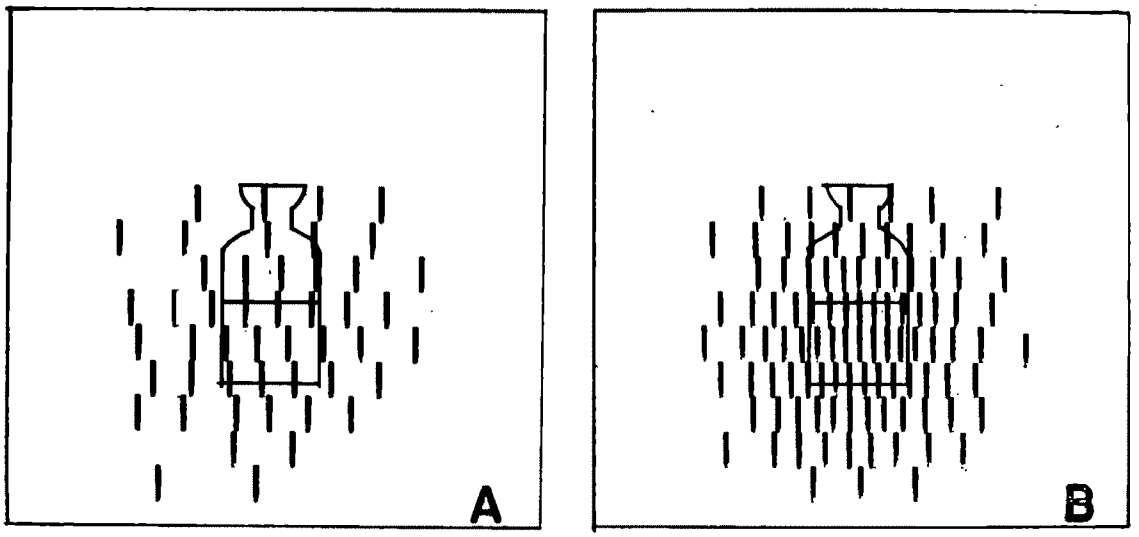


Fig. 10. Unbalance scans of 14 cc. "tumor" volume, 15/1 "tumor" to "brain" concentration. (*A*) 2 cm. left of midplane; (*B*) 4 cm. left of midplane, 1,100 volts.

TABLE I

POSITRON AND UNBALANCE RESULTS WITH DIFFERENT "TUMOR" TO "BRAIN" CONCENTRATIONS IN VARIOUS VOLUMES*

Volume of "Tumor"	μc Content	Concentration "Tumor"/"Brain"	Results on		
			Positron Scan	Unbalance Scan	
				Prints present at	
				right of center	left of center
8 cc.	.88	15/1	Discernible	Not Measured	
14 cc.	.88	8.7/1	Just barely discernible	5 cm.	1 cm.
42 cc.	.70	2.8/1	Not discernible	none	4 cm.
75 cc.	.88	1.6/1	Not discernible	none	4 cm.
8 cc.	1.8	30/1	Clear	2 cm.	1 cm.
14 cc.	1.8	15.4/1	Clear	2 cm.	1 cm.
40 cc.	1.4	5.6/1	Discernible	4 cm.	2 cm.
75 cc.	1.8	3.2/1	Discernible	3 cm.	1 cm.

* 1,000 volt photomultiplier setting; before correction for greater left sensitivity.

rior or posterior portion of the brain, the "tumor" phantom with a 15/1 concentration was moved forward 3 cm. from the center position in the sagittal plane. Scans were made with the "tumor" phantom in this position and to the right and the left of the midplane. These scans, both positron and unbalance, were essentially the same as for the "tumor" phantom located in the center of the brain and to the right and the left of center. The difference in the head thickness or absorber and scatterer did not affect the visible scans.

2. EFFECT OF "TUMOR"/"BRAIN" RATIO ON SCANS

"Tumor" phantoms of 8 cc., 15 cc., 42 cc. and 75 cc. volumes, each containing 0.88 μc of As^{74} diluted in water, were scanned. This was repeated with 1.8 μc of As^{74} under the same conditions. Table I shows the results of the positron and unbalance scans. These results can be summarized.

In order to get discernible positron scans with the scanners as set at this time: (1) if the volume is 10 cc., a "tumor"/"brain" concentration of 15/1 is needed; (2) if the volume is 15 cc., a 10/1 ratio is needed; (3) if the volume is 40 cc., a 6/1 ratio is needed; and (4) if the volume is 75 cc., a 3/1 ratio is needed.

Here as before, the unbalance scans gave an indication of the presence of lower concentrations than could be detected by the positron scans.

3. EFFECT ON SCANS OF A NONABSORBING "TUMOR" MASS

Water was put into new, uncontaminated "tumor" phantoms of different volumes. The same concentration of As^{74} was kept in the "brain" phantom as in previous scans. Positron and unbalance scans were made with the "tumor" phantom at the center and to the right and the left of the midplane.

Figure 11*A* shows the positron scan of 124 cc. volume. The "tumor" phantom could not be located in any of the positron scans. However, when the 124 cc. "tumor" phantoms were moved 2 cm. to the right, left-sided prints became visible, as in Figure 11*B*. As the phantom was moved further to the right, more left-sided prints were made (Fig. 11*C*). When the phantom was moved 4 cm. to the left, right-sided prints were produced (Fig. 11*D*).

As small a "tumor" phantom as 18 cc. could be detected if it were moved 2 cm. to the right or to the left.

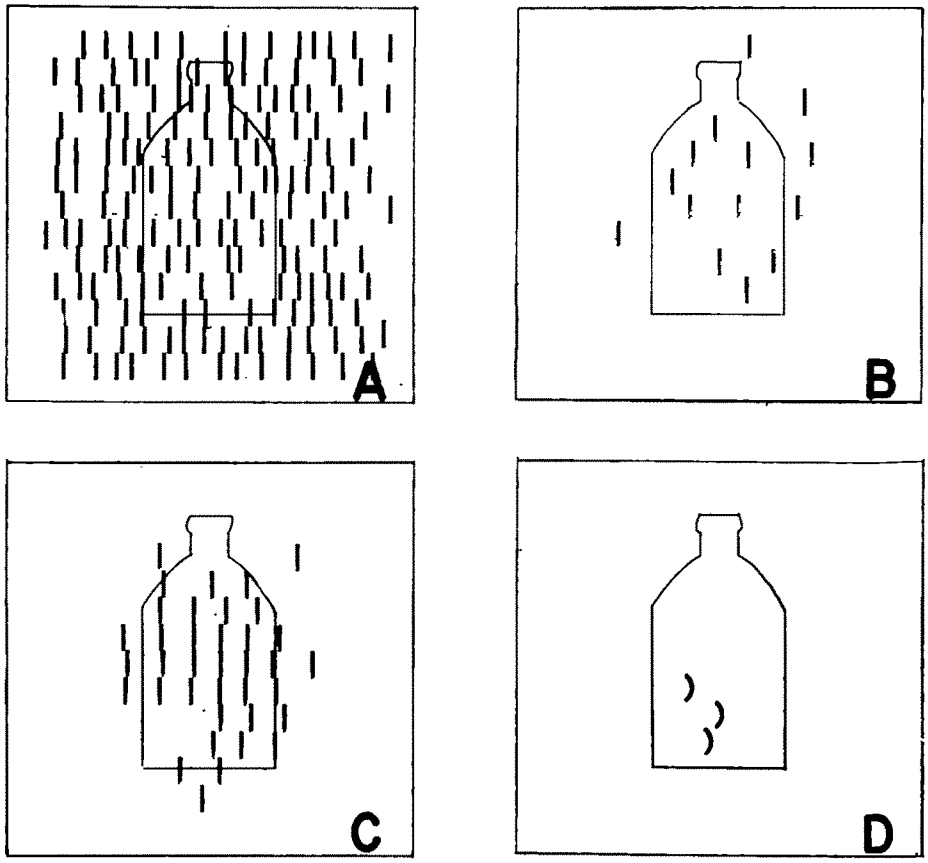


FIG. 11. Scans of 124 cc. "tumor" phantom filled with water (nonabsorbing mass). (*A*) Positron scan of "tumor" in the midplane; (*B*) unbalance scan of "tumor" 2 cm. right of midplane; (*C*) unbalance scan of "tumor" 4 cm. right of midplane; (*D*) unbalance scan of "tumor" 4 cm. left of midplane.

The "tumor" phantom was then moved 3 cm. anteriorly and scans were again repeated with the phantom in the midplane and to the right and the left. The results were essentially the same as for the central coronal plane.

Scans were also made with empty "tumor" phantoms. The results were the same as for the phantoms filled with water.

The implications of these scans where the tumor is "cold" are important and will be discussed later.

DIFFICULTIES ENCOUNTERED

As this investigation progressed, the causes for certain irregularities in the scans were found. Since others working in this field may have similar problems, we think it of value to present our findings.

1. SPURIOUS LEFT-HANDED PRINTS

The scanners were balanced with the spacer containing the test tube of As^{74} in the hole provided for this purpose (Fig. 12). No prints were made in a half hour of scanning, with the unbalance scanner set so that a difference of 1,500 counts would produce a print. However, when the "brain" phantom was scanned, left prints appeared in the anterior section of the scan. In order to make certain that the prints were not due to contamination of the outside of the phantom, the phantom was turned around 180 degrees. The scan was the same. Closer examination of the scanner detectors showed that even though the spacer was set in its "proper" place the detectors were not properly aligned. The right detector was angled too far forward. After a more

careful alignment, there were no more left-handed prints throughout the scan.

2. UNEQUAL SENSITIVITY OF SCINTILLATION CIRCUITS FOR DIFFERENT QUALITIES OF RADIATION

If the circuits were balanced for direct radiation, they were no longer in balance if scattering material was put on both sides of the As^{74} test tube. There were more left prints than right prints. Thus, the right and the left circuits had a different sensitivity for scattered radiation than for direct radiation. Since in actual scans of a patient's head the detectors receive both direct and scattered radiation, it was decided to balance the circuits with 7 cm. of masonite pressdwood of unit density on both sides of the As^{74} test tube inside the spacer.

Under these conditions, when the "tumor" phantom was the same distance to the right or the left, the right and the left-sided prints were approximately the same, with the center of the print configuration at approximately the center of the emitter.

A better way of balancing would be to make a phantom cube or ovoid with a hole in the center for the As^{74} test tube. However, the arrangement with the scattering material in the spacer is much better than one without.

3. RESPONSE TO VOLTAGE CHANGE

When we first started to use the scanner, the manufacturer suggested a setting of 1,000 volts across the photomultiplier tube. Part of this phantom work was carried out with this setting. Later this was changed to 1,100 volts. The sensitivity was markedly improved, so that where previously a 10/1 "tumor" to "brain" concentration had been necessary for a 30 cc. tumor to be seen clearly on a positron scan, now a 5/1 ratio sufficed to give good positron definition. The best voltage has yet to be found.

DISCUSSION

The smallest volume which was discernible on the positron scan under our optimum conditions of operation was 8 cc. with a "tumor" to "brain" concentration of 15/1. However, the ratio of actual tumor to brain concentrations as found by biopsy material[1] is far less than 15/1, and may vary from 3/1 to 10/1. If we assume a 5/1 ratio, the smallest tumor detectable on the positron scan would be about 30 cc. in volume.

The unbalance scanner is more sensitive. As small a phantom volume as 4 cc., with a 15/1 ratio of "tumor" to "brain" concentrations, can be detected if it is moved 2 cm. off center. We understand that some of the users of the positron annihilation scanners have not been using their unbalance

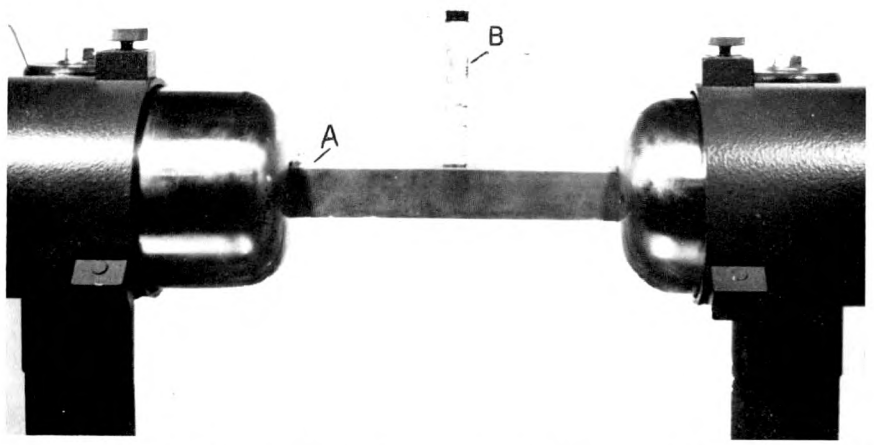


FIG. 12. Set-up of spacer for balancing the unbalance circuit. A—Spacer fitted into collimator openings; B—test tube containing As^{74}.

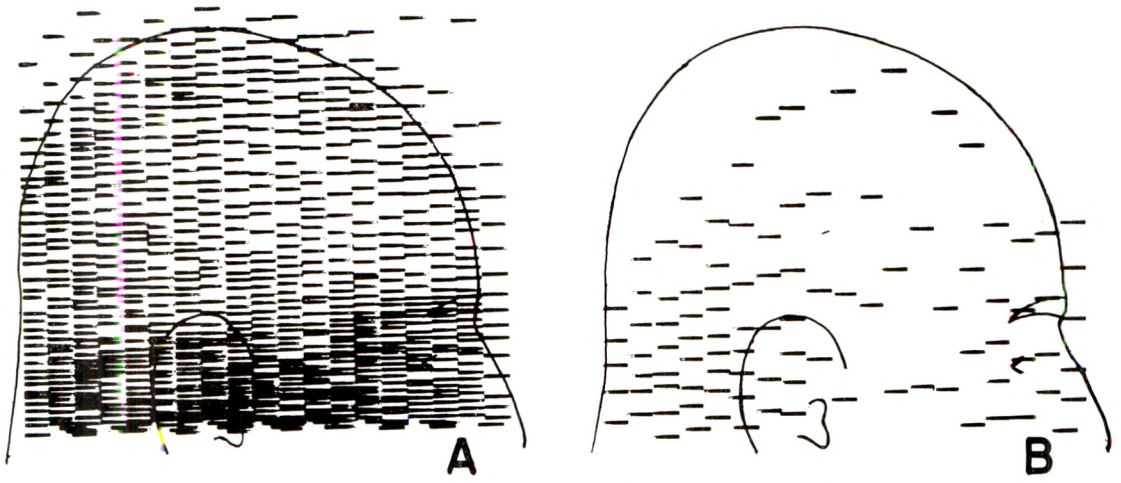


Fig. 13. Lateral view of a patient's head. (*A*) Positron scan. (*B*) Unbalance scan.

circuits because of difficulty in balancing. The unbalance scan yields information at lower concentrations and smaller volumes than the positron scan, and therefore these scans should be made.

When an abnormality appears on the positron scan, its location is much more precise than on the unbalance scan.

When there isn't any positron localization and either left or right prints are produced, a mass of material may be present which is not picking up As^{74} and therefore is in the opposite side from that indicated by the print. A left-sided print may mean that some "cold" material is present on the right side and vice versa. However, one must be aware that even with large volumes one needs a ratio of tumor to brain concentration of at least 3 to 1 to get a discernible positron print. Thus, unbalance prints without positron prints do not indicate whether the tumor mass is to the right or the left, but only where it is in the plane of scanning. Other information, either clinical or from scans of the head at 90° off the previous position, is necessary to localize the center of the lesion in the coronal plane. Unbalance scans do not define the lesion very accurately, for a small lesion to one side may give a large area of abnormal prints while a large lesion, toward the center, may give a limited area of abnormal prints.

INTERPRETATION OF BRAIN SCANS ON PATIENTS

There does not seem to be any easy way, short of using it under various conditions, to learn the best method of using an instrument. The use of a phantom gives information which is valuable in the interpretation of brain scans.

Positron scans of a patient in which the tumor is clearly defined are shown in Figures 2*A* and 3*A*. This patient was operated on and a meningioma was found in the exact location indicated in the positron scans.

Often no differentiation can be found on the positron scan, while the unbalance scan does give information (Fig. 13, *A* and *B*). The positron scan (Fig. 13*A*) does not indicate a lesion. However, the unbalance scan (Fig. 13*B*) shows a concentration of left-sided prints in the occipital region. This localizes the lesion approximately in the center of these prints. Left-handed prints without positron localization may mean a decreased uptake on the right side, or an increased uptake on the left side when the concentration is below that necessary to yield positron prints. Here the anteroposterior scans (Fig. 14, *A* and *B*) resolve this difficulty. On the positron scan (Fig. 14*A*) there is a slight increase in prints in the area above the left eye. The unbalance scan (Fig. 14*B*), which normally should be sym-

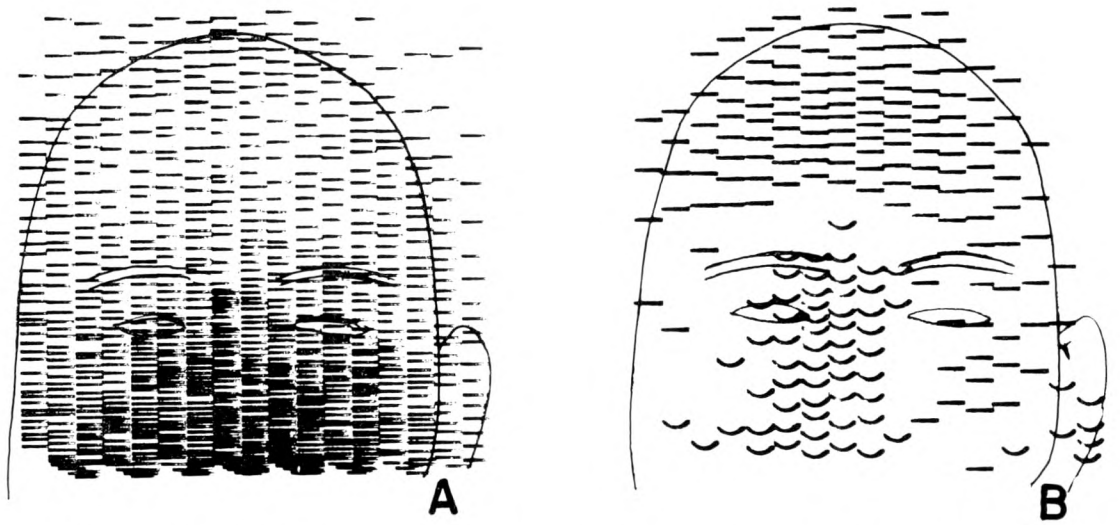


FIG. 14. Anteroposterior view of patient in Figure 13. (*A*) Positron scan. (*B*) Unbalance scan.

metric around the sagittal plane, shows on the left side of the face a missing portion of front (curved) prints, and then posterior (straight) prints, whereas on the opposite side of the face it shows a preponderance of front (curved) prints. The evidence from these two scans indicates that there is a lesion with greater uptake in the left posterior portion of the head. This, together with the unbalance scan shown in Figure 13*B*, locates a lesion of increased uptake in the left occipital portion of the head. This localization was confirmed by the neurosurgeon.

Figure 15, *A* and *B* shows the scans of a patient where the positron scan (Fig. 15*A*) did not show any definite lesion, but the unbalance scan (Fig. 15*B*) showed a predomi-

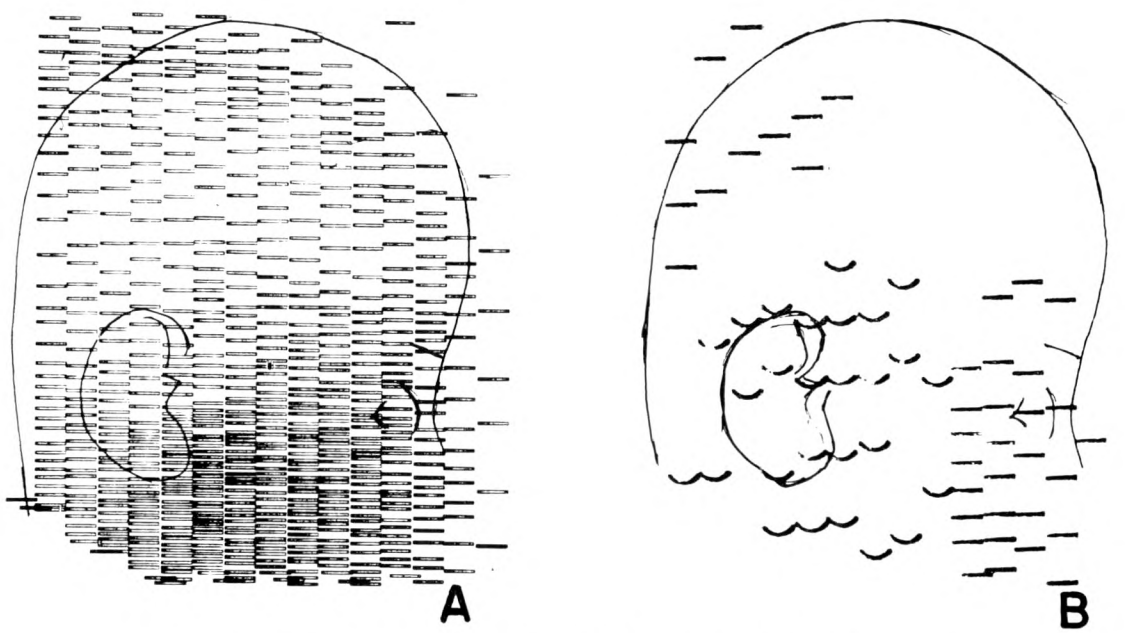


FIG. 15. Lateral view of a patient's head. (*A*) Positron scan. (*B*) Unbalance scan.

nance of right-sided prints in the region of the temporal lobe. This could be interpreted as an increase in uptake on the right side or a decrease in uptake on the left side. The child was unconscious and did not cooperate, so that an anteroposterior view could not be made. However, the neurologic examination indicated that there was something in the left hemisphere. The interpretation using the scan and clinical information was that there was a region of decreased uptake in the left temporal zone. Surgery was performed. The dura was incised and 60 cc. of cerebrospinal fluid were evacuated from the left temporal subdural space.

In the interpretation of scans, all available information should be used to get a picture in which all the pieces fit together into a coordinated whole.

SUMMARY

1. Positron annihilation and unbalance scans were made of a "tumor" in a "brain" phantom using a fixed concentration of As^{74} in the "brain" phantom while varying the volume, concentration and position of the "tumor" phantom.

2. Unbalance scanning is more sensitive than positron scanning. Unbalance prints appear when there is no discernible difference in the positron prints. In this study a 4 cc. volume with a 15/1 ratio of concentrations could be detected on the unbalance scan at 2 cm. to the right or the left of center. An 8 cc. "tumor" volume was needed to give a discernible positron scan.

3. The definition of the "tumor" phantom on the positron scan is good, while that on the unbalance scan is diffuse. There is equal definition on the positron scan, whether the "tumor" phantom is at the center or to the right or the left of the midplane.

4. A "cold" tumor, as simulated by having water in the "tumor" phantom, cannot be detected on the positron scan up to a 124 cc. volume, the largest investigated in this study, but does appear on the unbalance scan. Location of the "tumor" phantom to the right of the midplane appears as left-sided prints and vice versa.

5. An analysis of patient brain scans is given in terms of the findings in the phantom work.

6. The scans are very sensitive to the voltage across the photomultiplier tube. A "tumor" phantom of 8 cc. volume with a "tumor" to "brain" concentration of 15/1 is hardly discernible in a positron scan with a voltage setting of 1,000 volts, but is clearly seen at 1,100 volts.

7. A method of balancing the right and left circuits, by which the test tube of As^{74} is surrounded by unit density scattering material, simulates conditions for the head more closely than test tube alone.

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THE LOCALIZATION OF INTRACRANIAL SPACE-OCCUPYING LESIONS BY FLUOROBORATE IONS LABELLED WITH FLUORINE 18*

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THE physiologic behavior of fluoroborate ions has been investigated in previous studies.[1,2] It was found that fluoroborate ions accumulate in brain to a much smaller extent than in other tissues. In view of the limited permeability of fluoroborate through the blood brain barrier, it has been suggested to apply this ion labelled with positron emitting fluorine 18 as a tracer for the localization of space-occupying pathologic lesions in the brain. The advantages of positron emitters for this purpose have been previously emphasized and they have been so utilized.[12,13] Preliminary clinical tests on patients suffering from intracranial lesions gave promising results. These results were substantiated by a comparative study of various potential tracers carried out on rats.[3] Further experiments employing fluorine 18 labelled fluoroborate have confirmed the efficacy of this tracer for such applications.

PREPARATION OF LABELLED FLUOROBORATE

Fluorine 18 was prepared by irradiation of Li_2CO_3 in the Israel Research Reactor I, utilizing the $Li^6(n,\alpha)H^3$, $O^{16}(H^3,n)F^{18}$ nuclear reactions. An amount of 10 g. Li_2CO_3 of normal isotopic composition, sealed in a silica tube, was irradiated for 1 hour at a flux of 5×10^{12} n/cm.^{2}/sec. After irradiation, the ampule was crushed in a stainless steel beaker and the carbonate was dissolved in a slight excess of 6*N* HCl. One millimole of concentrated H_3PO_4 was added, followed by 100 mg. $CaCO_3$. The solution was then neutralized. The calcium phosphate which precipitated and carried with it the fluoride by coprecipitation was filtered off, washed and then dissolved in 6*N* HCl, and the acidity was adjusted to pH 0; 5 mg. NaF was then added followed by 100 mg. KBF_4. Complete isotopic exchange accompanied by partial hydrolysis[4] was obtained at room temperature in 10 minutes. The solution was then neutralized with sodium bicarbonate to pH 5.5 and passed through a column of chromatographic alumina for quantitative removal of fluoride ions. The labelled KBF_4 was obtained at a specific activity of over 10 μc/mg. The radiochemical purity was determined by following the decay of activity for 16 hours and the product was found radiochemically pure to 1 part in 10^5. The chemical purity was assayed by adding carrier fluoride and precipitating PbClF from the fluoroborate solution. The labelled fluoroborate was shown to contain less than 1 per cent labelled fluoride ions. As the potassium fluoroborate solution was ingested orally, no sterilization procedure was undertaken.

INSTRUMENTATION

The activity distribution in brain was determined by a coincidence scintillation measuring system. This consisted of one cylindrically collimated 1 inch sodium iodide crystal aligned within 1 degree with another shield but uncollimated 2 inch scintillator. The distance between both detectors was adjustable between 25 to 50 cm. without altering the alignment. Both scintillators were connected through twin preamplifiers, amplifiers and single channel analyzers (set between 400–550 kev.) to a

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slow coincidence analyzer (time of resolution, 10). The coincidence pulses were counted by a scaler with a preset timer. The random coincidence due to gamma background in a radiation field of 10^{-1} millirads per hour did not exceed 2 cpm.

The counting efficiency of a positron-emitting point source localized between the detectors was changed by a factor of four (distance between detectors 30 cm.) when the source was moved from the noncollimated to the collimated detector. This arrangement enabled the approximate location of positron emitting source by two measurements along the same line, interchanging the positions of the two detectors. A symmetric distribution of activity along the line of measurement gave the same value of counting rate in either position of the detectors. An increase in coincidence counts on interchanging the detectors indicated an uneven distribution along the line of measurement, with an increased activity at the proximity of the collimated detector.

MEASURING PROCEDURE

Two to three hundred microcuries of KBF_4^{18}, 10 μc/mg. in 50 ml. water were orally administered followed by a glass of tap water. The measurements of activity were started 120 minutes after ingestion.

The patient was seated with his head held in a fixed position. The aligned detectors were then placed at various points of the skull and the activity was measured twice along the same line, interchanging the positions of the detectors.

Eight lines of measurements were chosen:

1. Left supraorbital frontoparietal sagittal line
2. Supranasal frontoparietal sagittal line
3. Right supraorbital frontoparietal sagittal line
4. Left frontal and right parieto-occipital line (horizontal)
5. Right frontal and left parieto-occipital line (horizontal)
6. Left supraorbital fronto-occipital line 30°
7. Supranasal fronto-occipital line 30°
8. Right supraorbital fonto-occipital line 30°

The activity at each point was counted for 60 seconds, with the count repeated 2 to 3 times; the times of measurement were also recorded. The average values of these determinations corrected for radioactive decay were taken. The measurements at all 16 points took 35–45 minutes, after which the whole procedure was repeated as a check. A deviation of over 25 per cent from symmetric distribution of activity in the skull was taken as suggestive of a pathologic condition.

A typical case is presented in Figure 1. The values are given in counts per minute, with the collimator at the point indicated. All values presented are after correction for radioactive decay. A region of increased activity is suggested in the lower left hemisphere; this suggestion was confirmed by surgery and a glioma was found in the specified region.

RESULTS AND DISCUSSION

Over 50 patients have been examined for brain tumors using fluoroborate F^{18} as a tracer since 1958. Thirty-six of these were examined by the improved procedure described above. The results of the findings are presented in Table 1.

It can be seen that in cases of meningioma or glioma, as well as in the presence of other brain tumors, the fluoroborate tracer method gave satisfactory indications. In 16 out of 18 positive results, the localization of the lesions was confirmed within 5 cm.; in 2 cases, 1 meningioma and the other a lesion which caused unilateral exophthalmos, only the lateralization of the affected hemisphere was indicated. Tumors of the pituitary and of the third ventricle gave ambiguous results by the fluoroborate method, owing to the relatively higher normal accumulation of fluoroborate in the pituitary gland.[6,8]

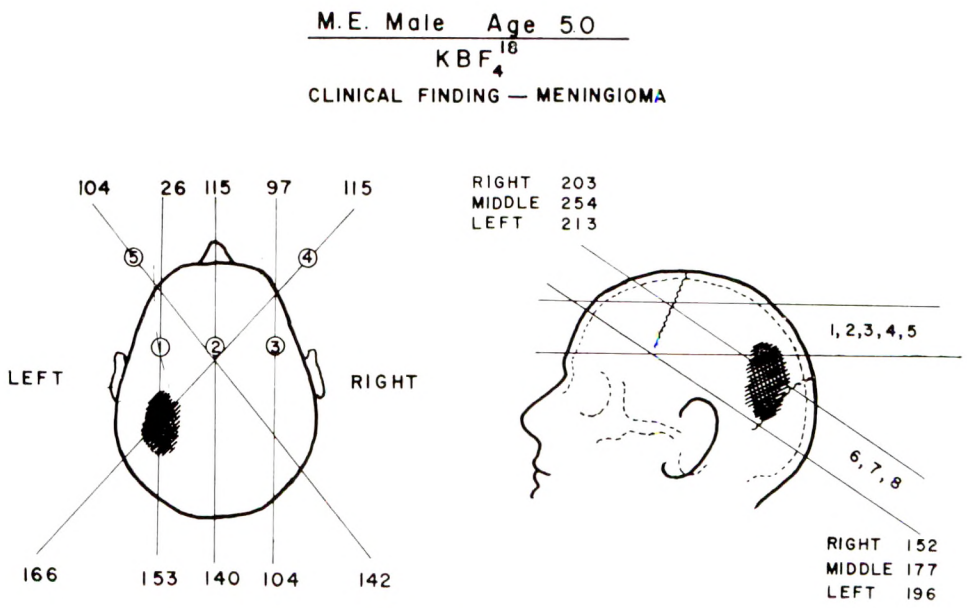


Fig. 1

The procedure is based on the detection of asymmetries in the activity distribution; any region in which activity was symmetrically distributed could escape detection. Vascular malformations gave positive results only when the blood content of the affected region was significantly different from normal tissue.

A good correlation was found between negative findings by the fluoroborate method and the corresponding pathologic findings. In all cases positive tracer results were clinically confirmed; only in 1 case the fluoroborate method gave a false negative result, when it failed to detect a glioma.

Table I

Correlation of space occupying intracranial lesions

Clinical and Surgical Findings	KBF_4^{18} Examinations	
	Positive	Negative
1. Meningioma	5	
2. Glioma	5	1
3. Vascular malformation	2	2
4. Tumor of third ventricle	1	1
5. Metastases of bronchus carcinoma	1	
6. Acusticus neuroma	1	
7. Exophthalmos	1	
8. Encephalitis		1
9. Tumor of the pituitary	1	3
10. Eosinophilic granuloma		1
11. No pathologic findings		9
12. Uncertain cases	1	

Several isotopes have been applied as tracers for the localization of space-occupying intracranial lesions. These include gamma emitters, *e.g.*, iodine 131, sodium iodide,[5] labelled serum albumine,[10,11] RISA, labelled iodo-fluorescein,[9,14] mercury 203 (labelled neohydrin[7]) and the positron emitters arsenic 74 (as sodium arsenate[12]) and copper 64 (labelled copper versenate[13]). It is evident that the annihilation radiation of positron emitters offers a better spacial resolution of the affected regions as compared with gamma emitters.[14]

These tracers were examined in a comparative study on rats[3] in which the parameters investigated were the blood/brain and muscle/brain specific activity ratios. It was assumed that the permeability of brain lesions is comparable to that of muscular tissue.[9]

The accretion in bone tissues was checked as well as the rate and route of excretion. Scanning of the brain *in vivo* is not applic-

Table II

COMPARISON OF VARIOUS TRACERS FOR THEIR PENETRABILITY INTO BRAIN TISSUE OF RATS

Relative Specific Activity	Time after Injection (min.)	Sodium Arsenate	Copper Versenate	Fluoro-Fluorescein	Potassium Fluoroborate
Blood / Brain	30 / 120	22 / 14	37 / 12	29 / 11	55 / 44
Muscle / Brain	30 / 120	5.7 / 4.0	4.2 / 8.3	1.5 / 1.2	3.6 / 6.4
Cranial bone / Blood	30 / 120	0.25 / 0.27	0.14 / 0.8	0.08 / 0.63	0.35 / 0.26
Blood at 30 min. / Blood at 120 min.		2.2	2.2	2.9	6.2
Per cent dose injected in intestines	120	36	32	18	2.6

able in the presence of high relative concentrations of tracer in bone.

The highest relative blood/brain specific activity ratios (Table II) are attained with fluoroborate. The muscle/brain ratios of fluoroborate are comparable to those of sodium arsenate and copper versenate.

Fluoroborate shows fast clearance from the organism which proceeds mainly via the kidneys in contrast to the other tracers examined. Fluoro-fluorescein, which has a higher molecular weight, shows a very limited permeability into muscle, and is expected to detect changes in the brain vascularization, rather than alterations in tissue permeability.

Fluorine 18 compares favorably in its nuclear properties with Cu^{64} and As^{74}. Since As^{74} is not a pure positron emitter (53 per cent positrons simultaneously with a 0.596 mev. gamma ray), a high degree of collimation and, consequently, high tracer doses are required to ensure good directional resolution. A single brain examination involves a dose of about 2.5 mc, resulting in a total body radiation dose of over 3 rads. Cu^{64} emits only 19 per cent positrons with a radiation dose of over 3 rads to the liver, which is the critical organ as regards the tumor of copper versenate.[14] As fluorine 18 is a pure positron emitter with a short half life (110 min.), a single examination using 300 μc, which is an adequate amount for the procedure adopted in this study, would expose the patient to a total body radiation dose of only about 10 millirads. Thus, the exposure risk involved in such an examination would be negligible. The only critical organ for fluoroborate is the thyroid gland, but the accumulation is negligible at doses higher than 0.1 mg./kg. body weight.

If the automatic scanning technique developed by Sweet *et al.*[13] were adopted for fluoroborate, the radiation dose to the patient would still be thirty times smaller than that delivered by arsenate. Moreover, the fluoroborate examinations, because of the short half-life, may be repeated after a few hours if needed to confirm the diagnosis.

SUMMARY

1. Potassium fluoroborate labelled with fluorine 18 has been applied as a tracer for the localization of intracranial space occupying lesions.

2. The distribution of radioactivity in brain was determined by a coincidence system along eight lines of measurement.

3. A satisfactory correlation was found between diagnosis with fluoroborate and other clinical findings.

4. A comparative study on rats demonstrated the efficacy of fluoroborate as a tracer for damage in the blood brain barrier.

5. The advantage of fluoroborate as a tracer over other tracers previously applied for brain scanning is discussed.

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A RADIOISOTOPE ACCOUNTABILITY SYSTEM*

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EXPANDING utilization of radioactive materials in the biologic sciences increases the concern about adequate records of the use and disposal of these materials. The United States Atomic Energy Commission regulations specify that records must be kept. Each radioisotope laboratory maintains these records but the format of the bookkeeping system varies widely from one laboratory to another.

After 8 years of trying different methods, the following system has proved highly effective, yet not cumbersome to manage. Simplicity and accuracy are its main features.

METHODS

A single entry method is used—the ledger (Table I) as the detailed record, with a consolidated record (Table II). Each isotope is identified by name, lot number, assay, and date of assay. The day the isotope is received, information from the container label is recorded in the ledger and in the consolidated record as of that date as explained in detail below. This label or a copy thereof is filed by the date of assay for future reference.

Three types of entries are made in the ledger; namely, receipt, usage, and disposal (Table I). The first entry represents the receipt of the isotope and the date it is received. The information from the isotope container label is recorded in Column 2, the total amount in millicuries in Column 8, and the total amount in milliliters in Column 9. The second type of entry, information gained from Table III, "User's Report," is recorded the same day any portion of the isotope is used. The date is placed in Column 1, patient's name in Column 3, the name of the test or experiment in Column 4, the amount in millicuries used in Column 6, and the amount in milliliters withdrawn in Column 7. The previous milliliter balance (Col. 9) minus the milliliters used (Col. 7) gives a new milliliter balance (Col. 9), and the product of this multiplied by the assay is the new balance to be entered in Column 8. In each entry the total of Column 6 and Column 8 subtracted from the previous entry of Column 8 reveals the amount of decay which is entered in Column 5. It is possible to know at all times the amount on hand, the millicurie strength of that amount, and the total amount of decay which has occurred from the date of receipt to the date of the last entry. This enables more effective utilization of radioisotopes. The third type of entry indicates the remaining amounts that are transferred from Columns 8 and 9 to disposal, as well as the date. The date is recorded in Column 1, "To Disposal," and the milliliter balance is written in Column 2. The millicurie balance is transferred to Column 5. The total of Columns 5 and 6 must equal the first entry in Column 8; *i.e.*, total decay plus total used equals total received in millicuries. The total of Column 2 (milliliters to disposal) and Column 7 (milliliters used) equals the first entry in Column 9—milliliters received. Another accuracy check is available. On any date, the line total of Columns 5, 6, and 8 equals the previous entry in Column 8.

A consolidated record (Table II) is maintained wherein a separate sheet is kept showing the total amounts received, used, and decayed for each radioactive material. The total amount of money spent for a specific isotope is also easily computed. The first entry in the consolidated record is made simultaneously with the first entry in the ledger; the name of the isotope in

* From the Radioisotope Service, Veterans Administration Hospital, Omaha, Nebraska.

TABLE I

LEDGER

1 Date	2 Isotope	3 Patient	4 Experiment or Test	5 mc Decay	6 mc Used	7 ml. or Caps Used	8 mc Balance	9 ml. or Caps Balance
11-6-61	I^{131} sodium iodide Lot #I-123-45 Assay 5.0 mc/ml. as of 11-6-61						20.000	4.00
11-6-61			Tetracycline (CE)	0.000	5.000	1.00	15.000	3.00
11-7-61		John Doe	Therapy (JH)	1.224	4.592	1.00	9.184	2.00
11-8-61			Tetracycline (CE)	2.041	7.000	1.66	0.143	0.34
11-8-61	To Disposal 0.34 ml.			0.143			0.000	0.00
	Total 0.34 ml.			3.408	16.592	3.66	0.000	0.00

TABLE II

CONSOLIDATED RECORD

1 Isotope	2 Lot No.	3 Date Received	4 Date of Completion	5 No. of Caps* Received	5 No. of Caps* Used	6 mc Decay	7 mc Used	8 mc Received
I^{131} sodium iodide solution	I-123-45	11- 6-61	11- 8-61			3.408	16.592	20.000
	I-234-56	11-10-61	11-14-61			2.383	7.767	10.150
(Cost								
10 mc/$16.50	I-345-67	11-20-61	11-20-61			0.000	20.636	20.636
20 mc/$29.00)	I-456-78	11-27-61						10.126

* This column used for capsule record.

TABLE III

USER'S REPORT

Name of Isotope	I^{131} sodium iodide
Lot No.	I-123-45
Assay	5.0 mc/ml.
Date of Assay	11-6-61
Assay Used	4.592 mc/ml.
Date Used	11-7-61
mc Removed	4.592 mc
ml. Removed	1.0 ml.
Patient's Name	John Doe
Experiment or Test	Hyperthyroidism Therapy
Used by	Robert Brown, M.D.

Column 1, lot number in Column 2, date received in Column 3, and the total amount in millicuries received in Column 8. When appropriate, the ledger account is closed, and the total amounts of millicuries used are then transferred to the consolidated record. The date of completion is recorded at this time in Column 4. Stock levels of isotopes are determined by considering the information in the consolidated record. Needs can then be easily anticipated. These records must balance with the disposition records maintained by the health physicist.

The consolidated record offers a review of

the complete history of each isotope in the laboratory with detailed information readily available in the ledger. This plan is equally adaptable to laboratories preparing their own isotopes. This system has been utilized for the past 22 months and has resulted in a 44 per cent increase in the number of studies accomplished with a 51 per cent decrease in the amount of isotopes purchased.

Richard E. Ogborn, M.D.
Veterans Administration Hospital
4101 Woolworth
Omaha 5, Nebraska

A DESIGN FOR A RADIOISOTOPE UNIT FOR A LARGE GENERAL HOSPITAL*

By K. H. CLARKE, M.Sc., A.R.C.S., A.Inst.P., *and* JEAN MILNE, F.I.R.
MELBOURNE, VICTORIA, AUSTRALIA

THE authors have had over 10 years' experience in the clinical application of radioisotopes and of recent years have been concerned with the design of 5 hospital radioisotope units, 3 of which have been in new buildings. Being associated also with the later operation of these radioisotope units, it has been possible to experience the advantages and disadvantages of different arrangements of facilities and to improve the layout of each succeeding design.

All the activities and movement of personnel of the radioisotope units have been studied including: (1) the handling of bulk supplies of radioisotopes and the breakdown into smaller doses; (2) the handling and preparation of very large therapeutic doses; (3) the preparation of special trays and trolleys for administration; (4) the handling of contaminated equipment following therapeutic procedures; (5) the handling of very radioactive excreta for radioactive assay; (6) the handling and measurement of tracer doses and other samples of low-level activity; (7) sterile radioactive procedures; (8) *in vivo* patient measurements; and (9) low-level decontamination. Consideration has been given to the sequence of steps in all procedures and to the location of the various work areas so that movement shall be minimal and all unnecessary cross-movement shall be avoided.

It is necessary, of course, to design for the future and to make provision for the accommodation of new equipment and additional staff that may become necessary as the volume of work increases and new techniques are developed.

THE RADIOISOTOPE UNIT

The latest design, shown in Figure 1, occupies 1,200 square feet and this is regarded as a reasonable minimum for a compact unit being planned for a new building. Such a unit could handle the routine tracer investigations (thyroid, renal, hematologic including the sterile labelling of plasma and red blood cells, scanning, etc.) and therapeutic administrations including the lengthy administration procedures such as are involved in the use of radioactive colloidal gold (Au^{198}) or radioactive colloidal phosphates (P^{32}) for the treatment of serous effusions.

The unit consists of a number of separate work areas with the "hot" or more radioactive areas shown on the left of the diagram and the less radioactive areas to the right, so designed as to cause no interference of the sensitive detecting equipment from the radioactive sources.

The High-level Preparation and Decontamination Area (A) includes a well protected store to contain radioactive materials and any highly radioactive excreta or waste, a reasonably large fume cupboard with double sinks for handling obnoxious or volatile materials, work benches for the preparation of large therapy doses and for the decontamination of highly radioactive equipment, a soiled linen receiver, and a washing machine and drying cabinet for the decontamination of bed and other linen prior to dispatch to the laundry.

The sink outlets from the fume cupboard and the adjacent work bench should be connected directly to a main outlet for the satisfactory dilution of liquid radioactive waste.

Space has also been allotted for a monitor trolley and for other equipment trolleys.

The Patient Administration Room (B) is immediately adjacent to the Preparation Area (A) in order to keep all highly radioactive sources to one end of the radioisotope unit. It has direct access to the cor-

* From the Physics Department, Cancer Institute, Melbourne, Victoria, Australia.

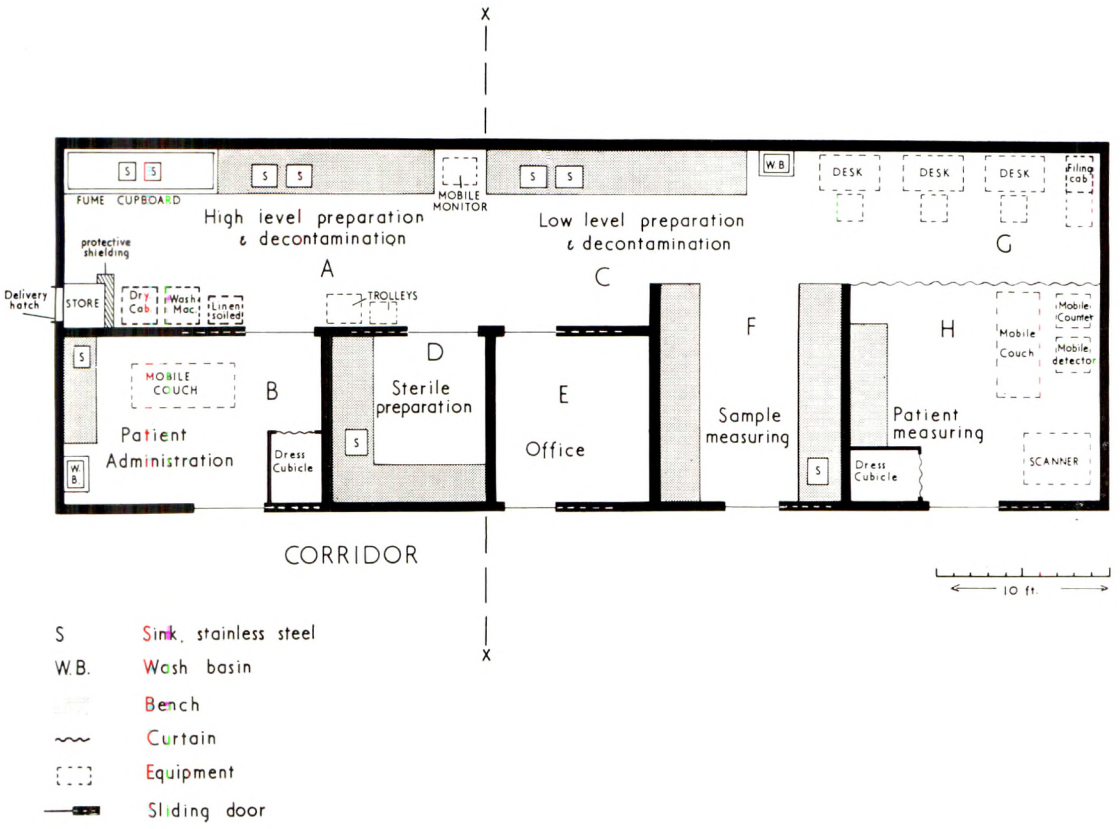


Fig. 1. Design of a Radioisotope Unit for a large General Hospital.

ridor and to the Preparation Area (A) in order to facilitate the movement of the dose and any special equipment required for administration as well as for the subsequent removal of contaminated equipment to the decontamination area. This Patient Administration Room (B) must be large enough to accommodate equipment, a couch and several persons (participants and students). It is envisaged that all therapeutic doses (oral, intravenous and intracavitary) and tracer doses which do not require immediate *in vivo* radioactive measurements could be administered in this room. Provided all administration equipment is handled from monitored stainless steel trays, there will be no danger of cross contamination.

Tracer tests which require immediate *in vivo* radioactive measurements may best be administered in the Patient Measuring Room (H).

The Low-level Preparation Area (C) lies in the center of the unit, giving easy access to the High-level Preparation Area (A) to facilitate the taking of aliquots from the very active stock solutions, doses or excreta, and also giving easy access to the Sample Measuring Area (F).

From experience it is recommended that "low-level" decontamination be performed in this area C in order to prevent the mixing of slightly contaminated equipment with grossly contaminated equipment, thereby facilitating the whole decontamination program.

Also in the center of the unit and adjacent to the Administration Room (B) is the Sterile Preparation Room (D) for the conduct of sterile procedures such as the labelling of red blood cells or plasma. This area should have a balance, centrifuge and incubator which are not used for any other purposes and a small refrigerator for the

storage of special doses. The room should be capable of complete isolation.

It is recommended that the Patient Measuring Room (H) should have as few permanent fixtures as possible. It is a great advantage to have the couch and counting equipment mobile and all that need be fixed are one work bench and the patient's dressing cubicle. The room is screened off from the Technician's Desk Area (G) by a curtain, so that if it is necessary to demonstrate a measuring technique to a large group of people the curtain can be drawn back, or if at some later date a measuring technique is adopted which involves more equipment than is currently used, then the room can be enlarged for the purpose.

An office (E) has been located within the unit and this could well be used for later expansion of facilities if such became necessary.

OTHER REQUIREMENTS AND FEATURES

The radioisotope unit must be located remote from roentgenographic machines, radioactive wards and other apparatus that could cause interference with the detecting equipment.

In the design of this unit, it has been assumed that a Patient Waiting Area and Patient Toilet Facilities would be available reasonably close to hand on the same floor. Also provision should be made for a shower-washroom nearby which could be used by staff in the event of a gross spillage of radioactive material. Such an event will be rare but, when gross spillage does occur, it is imperative that the person involved should wash the contaminated skin areas as soon as possible after the incident.

It is recommended that all work benches should have smooth nonporous surfaces with rounded corners and a minimum of joints for ease of cleaning, that sink areas be of stainless steel and that the inside walls of the fume cupboard should be painted with strippable paint for ease of decontamination if necessary.

Each sink should be fitted with a mixed hot and cold swivel outlet and a cold swan-neck outlet. The depth of each sink should be 9 inches and the distance from the mixed hot and cold outlet to the bottom of the sink should be 22 inches to facilitate handling of all laboratory equipment. Foot or elbow-controlled taps are recommended on all taps to permit operation without having to use potentially contaminated gloved hands.

Cupboards can be fitted beneath the benches with knee-spaces, or above the benches as required, and a number of nests-of-drawers with various depths of drawers has been found most useful.

The doors to the various areas are shown as sliding doors as these allow more space and avoid the hazards of swing doors when radioactive materials are being transported.

If the "hot" end of the unit can be located adjacent to a lift lobby, a hatch can be inserted in the wall of the radioactive store to permit the delivery of the radioactive material so as to avoid this material being carried through or past the laboratory.

If for any reason it is necessary to locate the unit in two nearby areas, the design given in Figure 1 can be broken along the line XX without gross loss of convenience and, if preferred, the sterile preparation room D and the office E can be interchanged.

If the use of the hospital radioisotope unit is entirely for tracer work, as might be the case in a Children's Hospital, then the area of 700 square feet to the right of line XX (Fig. 1), with the sterile preparation room replacing the office, will meet these requirements.

SUMMARY

The layout of the Radioisotope Unit presented is based on considerable experience and is designed to handle all the routine tracer investigations and radioactive therapy administrations of a large General Hospital.

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VOL. 88, NO. 2

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Sixty-third Annual Meeting: Shoreham Hotel, Washington, D. C., October 2–5, 1962.

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Forty-fifth Annual Meeting: Mark Hopkins Hotel, San Francisco, Calif., April 1–4, 1963.

EDITORIALS

INSTRUCTION COURSES OF THE SIXTY-THIRD ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

PRESIDENT-ELECT Earl Barth, acting in his capacity as Chairman of the Program Committee of the 1962 Annual Meeting of the American Roentgen Ray Society, has requested that the Instruction or Refresher Courses be continued as an integral part of the educational aspects of the coming meeting in Washington, D. C. This type of teaching program, available to guests and graduate students as well as members, was first tried in 1939. The courses have been so well received that each succeeding president-elect has followed the pattern set by Dr. Edward Jenkinson in 1939.

A complete list of the faculty participating in this year's program, together with a brief abstract of each course, will be found in the succeeding pages of this issue of the JOURNAL. A registration blank is included and it is advisable to register in advance by mail.

Thirteen courses will be presented each afternoon but it will be possible to attend only one course each day. Most of the rooms are rather small and consequently it is quite likely that some courses will be completely sold out by mail.

Forty of the fifty-two periods will deal with diagnostic radiology and twelve with therapeutic radiology, physics, radiation biology and isotopes. In the therapy section there will be courses of a very practical nature dealing with treatment of malignancies of the skin, cervix, breast, and head and neck as well as courses in basic physics and radiation biology and clinical dosimetry. Problems and pitfalls in supervoltage therapy will come under discussion as will the ever interesting association and correlation of radiation therapy and chemotherapy. Two isotope courses will be offered, both dealing with diagnostic uses of isotopes.

Most of the major areas of present day interest in diagnostic radiology will be presented. In the field of techniques and equipment, courses will be given dealing with image amplification and cine studies. Special procedures will be covered in such areas as lymphography, mammography, arthrography, angiocardiography, bronchography and neuroradiology. Many courses are available which cover the everyday and the unusual problems encountered in gastrointestinal work, chest radiology, bone and joint examinations, pediatric radiology and sinus and mastoid studies.

The total number of courses presented is smaller than last year but the variety of topics and excellence of Instructors should make this an outstanding educational experience for us all and we are again very grateful to the distinguished faculty who give so freely of their time and talent.

HAROLD O. PETERSON, M.D.
Director, Section on Instruction



STANLEY A. CLARK, M.D.
1877-1962

STANLEY A. CLARK died at his home in South Bend, Indiana, on April 17, 1962, after a long illness. He was born at Galien, Michigan, on July 14, 1877, and attended school there.

Doctor Clark received his medical degree from Hahnemann Medical College in Chicago in 1898. He entered general practice in Galien at the age of 21. Following two years of practice there, he moved to South Bend in 1901 and installed the city's first x-ray unit.

In 1911, Doctor Clark went to Vienna to study radiology and after returning to

South Bend, he installed new x-ray equipment and offered free chest x-rays for suspected tubercular patients. He was instrumental in obtaining a new state law which led to the construction of Healthwin Tuberculosis Hospital and was on the Board of Governors for 43 years. Doctor Clark also installed the first x-ray department at Memorial Hospital of South Bend in June of 1912, and this department is still owned and administered by his associates. He served as a Captain in the Medical Corps in World War I.

Doctor Clark's membership in the American Roentgen Ray Society dates from 1902. He was also certified as a Diplomate of the American Board of Radiology, was a Fellow of the American College of Radiology, a member of the Radiological Society of North America, the American Medical Association, and the Indiana State Medical Association.

Doctor Clark's contributions to medicine were primarily through organizational and philanthropic channels, and these were many and of growing historical importance. He was one of the organizers and a long-time officer of the South Bend Medical Foundation, a non-profit pathological laboratory which serves the entire area. Doctor Clark was also one of the founders and directors of the Saint Joseph County Cancer Society, Chief of Staff and member of the Board of Directors of Memorial Hospital for many years, and a heavy contributor to the hospital financially.

Doctor Clark's business interests were equally extensive and important. In 1934 he helped reorganize and form the National Bank and Trust Company and served as president for ten years. He was also a director of the Associates Investment Company for 34 years.

Noted for his contributions to charities and philanthropic projects, Doctor Clark was responsible for the Health Center of the Boy Scout Camp and initiated the building fund for the Galien High School in 1947 which resulted in a 30 room building. Later, he donated the money for the school's cafeteria, kindergarten room, and physics laboratory.

On June 19, 1956, civic leaders and friends of Doctor Clark organized a community testimonial dinner in his honor, "A man whose determined effort and support have meant much to the communities in which he has lived."

On the day following Doctor Clark's funeral, an editorial appeared in the *South Bend Tribune*, which said in part:

"Some men by their deeds and basic humanitarian spirit fashion during their lives the monument by which they are remembered in death. Such a man was Doctor Stanley A. Clark of South Bend, who died this week."

Doctor Clark is survived by his wife, the former Flora Pollock.

Lester G. Ericksen, M.D.

Vol. 88, No. 2

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*
Donn G. Mosser, M.D., *Associate Director*

Titles and Abstracts of Courses Offered
Sixty-Third Annual Meeting
Shoreham Hotel, Washington, D. C.
October 2 to October 5, 1962

The Instruction Courses for the Sixty-Third Annual Meeting of the American Roentgen Ray Society will be held on Tuesday, Wednesday and Thursday afternoons, October 2, 3 and 4 at 3:00 P.M. and on Friday afternoon, October 5, at 1:30 P.M. Each course period will be of 90 minutes' duration and no other official activity will be scheduled for this time.

A special registration desk for these courses will be set up in the lobby of the Shoreham Hotel near the general registration area. Complete information concerning the courses as well as tickets for those courses still available can be obtained Monday afternoon, October 1, from 3:00 P.M. to 5:00 P.M. and daily, Tuesday through Friday, October 2–5, from 8:30 A.M. to 4:30 P.M.

Thirteen courses will be given each day during the four days of the meeting. A brief abstract for each course will be found on the following pages as well as a complete list of the faculty and the registration forms.

Admission to all courses will be by ticket. To avoid the confusion usually present on the first day of the meeting, tickets will be mailed to all those whose registration forms (Pink Sheets) reach the office of the Director before September 18, 1962. The envelope containing the tickets will be labelled in a conspicuous manner. Please be on the lookout for your tickets and remember to bring them to the meeting. Tickets will be issued in the order that the requests are received but the first mailing will be shortly after September 1.

How to Register and Obtain Tickets

After reviewing the abstracts of the available courses on the following pages, fill out the pink order sheet. Please make three choices for each day and include the course number and name of Instructor for each choice. A $2.00 registration fee is required from non-members for each course attended. Since it is possible to attend only one course a day for four days, the maximum fee is $8.00. Members of the American Roentgen Ray Society and graduate students or residents in Radiology are not required to pay for these courses but must fill out the pink order sheet indicating their choices.

Prior to September 16 please mail these order sheets to Dr. Harold O. Peterson, 1995 West County Road "B," St. Paul 13, Minnesota. After September 16 mail should be sent to Dr. Harold O. Peterson, Director of Instruction Courses, American Roentgen Ray Society, Shoreham Hotel, Washington, D. C.

THE FACULTY

1. Kurt Amplatz, M.D., Assistant Professor of Radiology, University of Minnesota Medical School, Minneapolis, Minnesota.
2. J. Robert Andrews, M.D., Chief, Radiation Branch, National Cancer Institute, Department of Health, Education and Welfare, Bethesda, Maryland.
3. Robert P. Barden, M.D., Department of Radiology, Chestnut Hill Hospital; Associate Professor, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.
4. John A. Campbell, M.D., Chairman, Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.
5. Chahin Movses Chahbazian, M.D., Associate Radiotherapist; In charge of Radiobiology, Penrose Cancer Hospital, Colorado Springs,

Colorado; Postgraduate Lecturer, University of Colorado Medical Center, Denver, Colorado.

6. John R. Cunningham, Ph.D., Clinical Physicist, Ontario Cancer Institute, Toronto, Ontario, Canada.

7. Gerald D. Dodd, M.D., Clinical Professor of Radiology, Assistant Radiologist, Jefferson Medical College Hospital, Philadelphia, Pennsylvania.

8. Lucille A. Du Sault, Radiation Physicist, Henry Ford Hospital, Detroit, Michigan.

9. Robert L. Egan, M.D., Department of Radiology, Methodist Hospital, Indianapolis, Indiana.

10. Bernard S. Epstein, M.D., Chief, Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York; Clinical Professor of Radiology, Albert Einstein College of Medicine, New York, New York.

11. Lewis E. Etter, M.D., Professor of Radiology, Falk Clinic, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania.

12. William R. Eyler, M.D., Chairman, Department of Radiology, Henry Ford Hospital, Detroit, Michigan.

13. Benjamin Felson, M.D., Professor and Director, Department of Radiology, University of Cincinnati, Cincinnati, Ohio.

14. Leo S. Figiel, M.D., Department of Radiology, The Grace Hospital, Detroit, Michigan.

15. Steven J. Figiel, M.D., Department of Radiology, The Grace Hospital, Detroit, Michigan.

16. Harry W. Fischer, M.D., Associate Professor of Radiology, Department of Radiology, University Hospitals, State University of Iowa, Iowa City, Iowa.

17. Robert H. Freiberger, M.D., Assistant Professor of Clinical Radiology, Cornell University; Department of Radiology, The Hospital for Special Surgery, Margaret M. Caspary Clinic, New York, New York.

18. Milton Friedman, M.D., Director, Radiation Therapy Department, Hospital for Bone and Joint Diseases; Professor of Clinical Radiology, New York University College of Medicine, New York, New York.

19. William E. Gannon, M.D., Department of Radiology, College of Medicine, State University of New York, Brooklyn, New York.

20. Brit B. Gay, Jr., M.D., Department of Radiology, Emory University, Atlanta, Georgia.

21. Roy R. Greening, M.D., Professor of Radiology, Assistant Radiologist, Jefferson Medical College Hospital, Philadelphia, Pennsylvania.

22. John W. Hope, M.D., Director, Department of Radiology, The Children's Hospital of Philadelphia; Professor of Clinical Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

23. Melville L. Jacobs, M.D., Chairman, Department of Radiology, City of Hope Medical Center, Duarte, California.

24. Harold G. Jacobson, M.D., Head of the Division of Diagnostic Radiology of Montefiore Hospital; Professor of Clinical Radiology, New York University College of Medicine; Senior Consultant in Radiology to the Veterans Hospital in the Bronx, New York, New York.

25. E. C. Klatte, M.D., Associate Professor of Radiology, Chief of Pediatric Radiology, Indiana University Medical Center, Indianapolis, Indiana.

26. Morton M. Kligerman, M.D., Director of Radiology, Grace-New Haven Community Hospital; Professor and Chairman of Radiology, Yale University School of Medicine, New Haven, Connecticut.

27. Jack S. Krohmer, Ph.D., Associate Professor of Radiology (Radiation Physics and Radiobiology), University of Texas Southwestern Medical School, Dallas, Texas.

28. Richard G. Lester, M.D., Professor and Chairman, Department of Radiology, Medical College of Virginia, Richmond, Virginia.

29. Alexander S. Macmillan, Jr., M.D., Director, Department of Radiology, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

30. Richard H. Marshak, M.D., Associate Radiologist, Mt. Sinai Hospital, New York, New York.

31. John G. McAfee, M.D., Associate Professor, Department of Radiology, The Johns Hopkins Hospital, Baltimore, Maryland.

32. William T. Meszaros, M.D., Director of Radiology, Illinois Masonic Hospital; Attending Radiologist, Cook County Hospital; Assistant Professor of Radiology, Northwestern University Medical School, Chicago, Illinois.

33. Russell H. Morgan, M.D., Radiologist-in-Chief, The Johns Hopkins Hospital, Baltimore, Maryland.

34. Walter T. Murphy, M.D., Director of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo, New York.

35. Sidney W. Nelson, M.D., Professor and Chairman, Department of Radiology, The Ohio State University Medical School, Columbus, Ohio.

36. Charles M. Nice, Jr., M.D., Professor and Chairman, Department of Radiology, Tulane University School of Medicine, New Orleans, Louisiana.

37. Robert S. Ormond, M.D., Department of Radiology, Henry Ford Hospital, Detroit, Michigan.

38. Paul A. Riemenschneider, M.D., Professor of Radiology, State University of New York, Upstate Medical Center; Director, Department of Radiology, Syracuse Memorial Hospital, Syracuse, New York.

39. Rieva Rosh, M.D., Associate Professor of Radiology, New York University School of Medicine; Visiting Radiation Therapist, Bellevue Hospital, New York, New York.

40. Philip Rubin, M.D., Chief and Professor, Division of Radiotherapy, Department of Radiology, University of Rochester, Strong Memorial Hospital, Rochester, New York.

41. Maurice D. Sachs, M.D., Associate Professor of Radiology, Western Reserve University, School of Medicine; Chief of Radiology, Veterans Administration Hospital and Forest City Hospital, Cleveland, Ohio.

42. Julian A. Salik, M.D., Radiologist-in-Chief, Sinai Hospital of Baltimore, Baltimore, Maryland.

43. William H. Shehadi, M.D., Director, Department of Radiology, United Hospital, Port Chester, New York.

44. Edward B. Singleton, M.D., Director, Department of Radiology, St. Luke's and Texas Children's Hospitals, Houston, Texas.

45. William J. Tuddenham, M.D., Professor of Radiology, University of Pennsylvania; Director of Radiology Department, Pennsylvania Hospital, Philadelphia, Pennsylvania.

46. Sidney Wallace, M.D., Senior Resident in Radiology, Jefferson Medical College Hospital, Philadelphia, Pennsylvania.

47. H. Stephen Weens, M.D., Professor and Head, Department of Radiology, Emory University School of Medicine, Atlanta, Georgia.

48. Marvin M. D. Williams, Ph.D., Professor of Biophysics, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

49. Robert E. Wise, M.D., Department of Radiology, The Lahey Clinic, and New England Baptist Hospital, Boston, Massachusetts.

50. Martin H. Wittenborg, M.D., Children's Hospital; Assistant Clinical Professor, Harvard Medical School, Boston, Massachusetts.

DESCRIPTION OF COURSES

COURSE 101
and
COURSE 201

Tuesday and Wednesday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

Course 101. 1. Technique, including a discussion of the various barium preparations. 2. Regional enteritis. 3. The malabsorption syndrome, including sprue, lymphosarcoma and Whipple's disease. 4. Problems in the interpretation of functional changes. 5. Inflammatory lesions of the duodenum, excluding duodenal ulcers.

The Small Intestine and Colon

Course 201. A-1. Infarction of the small bowel. 2. Tumors. 3. Collagen diseases.

B-1. Inflammatory lesions of the colon.

COURSE 102
and
COURSE 202

Tuesday and Wednesday

JOHN W. HOPE, M.D.
Philadelphia, Pennsylvania

Abdominal Mass Lesions in Infants and Children

Course 102. Nephroblastoma and neuroblastoma are the two most common abdominal mass lesions of infants and children. These two tumors, in their various manifestations, will be discussed as well as the differential diagnosis of each. Time will also be devoted to the therapy of each lesion. Most of the course will deal with these two entities, but the other miscellaneous and often very bizarre abdominal mass lesions of infants and children will likewise be considered.

Mediastinal Mass Lesions in Infants and Children

Course 202. Mass lesions of the mediastinum can be diagnosed with a high degree of accuracy simply by knowing whether they are situated in the front, in the middle or in the back. This course will attempt to give a comprehensive discussion of most of the mass lesions which occur in the mediastinum. There

are a few differences between the younger age group and the older age group and these differences will be discussed.

COURSE 103

Tuesday

RUSSELL H. MORGAN, M.D.
Baltimore, Maryland

Screen Intensification and Cineradiography

This course will deal with the physics and technical problems associated with screen intensification and cineradiography. The discussion will consider those aspects of x-ray image formation and perception which are important in the fields of screen intensified fluoroscopy and motion picture recording. Particular attention will be given to the practical problems associated with the use of various intensifier and cineradiographic systems in clinical radiology.

COURSE 104

Tuesday

WILLIAM T. MESZAROS, M.D.
Chicago, Illinois

Roentgen Cardiology of Adults

1. The normal heart
2. Cardiac mensuration
3. Cardiac chamber enlargement
4. Hypertensive heart disease
5. Rheumatic heart disease
6. Arteriosclerotic heart disease
7. Luetic heart disease
8. Cor pulmonale
9. Miscellaneous disorders: a. thyroid heart disease; b. anemia; c. hemachromatosis; d. Addison's disease; e. dissecting aneurysm; f. scleroderma; g. carcinoid
10. Diseases of the pericardium
11. Congenital heart disease in adults
12. The lungs in heart disease

COURSE 105

Tuesday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

New Concepts in Radiographic and Interpretative Techniques

This course consists of an analysis of the sources of error in roentgen diagnosis, a critique of existing concepts of "film quality," and proposals for modified radiographic and interpretative techniques designed to increase the accuracy of the method.

The inadequacy of "contrast" and "unsharpness" as criteria for evaluating the diagnostic usefulness of roentgenograms will be discussed and new criteria will be presented.

The significance of physical factors said to limit the "quality" of roentgenograms will be considered and the indications for so-called "high-kilovoltage" and "supervoltage" radiography as well as for the use of the "micro-focus" roentgen-ray tube will be specifically noted.

Psychophysiologic factors which limit the accuracy of roentgen diagnosis will be emphasized, and recent experimental studies relating to problems of visual search and image organization will be presented. Also, control of view-box and "surround" illumination, control of glare, and the selection of reading lenses—practical aids for the radiologist in perceiving critical "threshold" images—will be presented.

Pertinent experimental findings will be cited, but the practical clinical application of these concepts will be emphasized through the presentation of illustrative diagnostic roentgenograms.

COURSE 106
and
COURSE 206

Tuesday and Wednesday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses

Course 106. Description of standard techniques and positions with illustrative slides for roentgen examination of the skull and paranasal sinuses. Comparison of advantages of one projection over another with respect to visibility of particular structures. General considerations with reference to the cerebral and facial components of the cranium and various skull types. Analysis of individual bone components in producing the various lines and shadows seen in plain roentgenograms of the skull with examples of pathologic alteration of these normal structures. Extension of the analysis to the study of the sphenoid, frontal, temporal, ethmoid, parietal and occipital bones in various projections.

Course 206. Detailed roentgen anatomic analysis of the paranasal sinuses, middle ear, and mastoid processes with description of precision roentgenographic techniques employed. Study of the roentgen features of the middle cranial fossa and their relation to the sphenoid sinus. Consideration of the orbits and optic foramina with special attention to the frontal, ethmoid and sphenoid bone components in various projections as well as the features contributed by the zygomatic bones. Examples of pathologic alteration of these normal structures will be given. In conclusion, the maxillofacial components of the

cranium will be examined, including the lacrimal canals and soft tissue delineation.

COURSE 107

Tuesday

H. STEPHEN WEENS, M.D.
Atlanta, Georgia
BRIT B. GAY, Jr., M.D.
Atlanta, Georgia

Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes

I. Methods of roentgenologic study of pulmonary circulation: (a) roentgenoscopy with image amplification, (b) routine roentgenography, (c) laminagraphy, (d) pulmonary angiography and (e) cineroentgenography.

II. Basic function of pulmonary circulation: normal pulmonary arterial and venous pressure; normal pulmonary blood flow.

III. Increased pulmonary blood flow. Definition. Etiology: left to right shunts; admixture lesions. Roentgenologic signs. Pulsations of the pulmonary arteries in increased pulmonary blood flow.

IV. Decreased pulmonary blood flow. Definition. Etiology: left to right shunts with pulmonary stenosis or atresia. Roentgenologic signs. Roentgenologic appearance of collateral circulation to the lungs through bronchial arteries or anomalous arteries. Pulsations of pulmonary arteries in decreased pulmonary blood flow. Special consideration of pulmonary vasculature changes and pulsations in isolated pulmonary valvular stenosis.

V. Pulmonary venous hypertension—pulmonary edema. Roentgenologic signs. Significance of Kerley's (septal) lines. Special consideration of pulmonary venous changes in mitral stenosis. Importance of minimal signs of pulmonary interstitial edema as an indication of acute myocardial infarction.

VI. Pulmonary arterial hypertension. Definition. Etiology. Roentgenologic signs. Pulsations of pulmonary arteries in pulmonary hypertension.

The roentgenologic changes of the pulmonary circulation will be illustrated by cinefluorograms.

COURSE 108

Tuesday

ROBERT L. EGAN, M.D.
Indianapolis, Indiana

Over Six Years' Experience with Mammography

Over six years' experience has been gained with soft tissue roentgenography of the breast in a tumor institute with an active breast service. These studies have proved a highly accurate and valuable adjunct to the clinical and pathologic evaluation of breast lesions.

A simple and readily adaptable roentgenographic technique will be presented. Cases illustrative of typical benign and malignant disease will be shown and the diagnostic features of each described and tabulated. Differential diagnosis, sources of error, limitations of the procedure and the clinical and pathologic implications of the method will be discussed.

The results of a statistical analysis of the first 1,000 consecutive examinations (all with a follow-up from one to five years) will be reviewed. The error in 245 cases of malignant disease was 0.82 per cent.

COURSE 109
and
COURSE 209

Tuesday and Wednesday

ALEXANDER S. MACMILLAN, Jr., M.D.
Boston, Massachusetts

Course 109. **Sinuses**
Course 209. **Mastoids, Lateral Neck and Larynx**

COURSE 110
and
COURSE 210

Tuesday and Wednesday

SIDNEY W. NELSON, M.D.
Columbus, Ohio

Bronchography

Course 110. The radiologist who can properly perform and interpret bronchograms is in a position where he can make outstanding contributions to the diagnosis and understanding of many chest diseases. The proper therapy for any disease which affects the bronchial tree is often dependent upon the accurate determination of the nature and extent of the bronchial involvement. Inasmuch as the interpretation of bronchograms requires considerable knowledge of the normal anatomy of the tracheobronchial tree and some of the common anatomic variations, the pertinent anatomy will be discussed and illustrated. A good knowledge of bronchial anatomy will enable the radiologist to systematically identify each of the major segmental bronchi, because of his knowledge of the expected distribution of each of these bronchi. The identification of each of the major segmental bronchi is extremely important in interpretation.

Once the basic bronchographic anatomy has been considered, the details of the bronchographic technique will be described. This will include the method of performing topical anesthesia, introduction of intranasal catheter, method of injecting contrast medium in order to assure systematic filling of the desired segmental bronchi, proper positioning of patient for filling of each segment, and the spot filming

techniques employed to thoroughly record the filling of the desired segmental bronchi. This portion of the discussion will also include a description of some of the characteristic features of various contrast materials. The advantages of fluoroscopically controlled spot filming in comparison with conventional large film techniques will be emphasized. Good bronchographic technique can be mastered with little effort and can be quickly performed by the radiologist and his technical staff, thus precluding the necessity for resorting to the "team" approach which is often so time-consuming and frustrating in large busy hospitals.

Course 210. During the second session, the majority of the different types of bronchopulmonary pathology will be discussed including the following: 1. Chronic bronchitis: a. spasm; b. excessive secretions; c. dilated mucosal glands; d. destruction of mucosa. 2. Emphysema. 3. Segmental bronchiectasis. 4. Cystic lesions. 5. Bronchogenic carcinoma. 6. Pulmonary "coin" lesions. 7. Bronchial adenoma. 8. Erosion of bronchi by adjacent calcified lymph nodes. 9. Inflammatory strictures. 10. Bronchial relocation following atelectasis of surgical removal.

The value of bronchography in the differential diagnosis of many bronchopulmonary diseases will be emphasized throughout the course, as for instance, atelectasis due to bronchogenic carcinoma or other intrabronchial lesions. Segmental bronchiectasis with associated chronic pneumonic changes may mimic neoplasms on plain chest roentgenograms, but can be clearly identified by bronchographic methods. Chronic granulomas and other infections can frequently be identified by virtue of the bronchiectatic changes produced in adjacent bronchi, thus helping to differentiate these diseases from neoplastic processes. Certain pulmonary diseases have very specific bronchographic appearances; *e.g.*, bronchial adenomata, polycystic lung, etc.

It is hoped that this presentation will encourage radiologists to perform this extremely useful roentgenographic procedure more frequently in the future. In so doing, they will be able to make more significant contributions to the diagnosis and treatment of the patients referred to them for study by their colleagues who are not radiologists.

COURSE 121

Tuesday

LUCILLE A. DU SAULT
Detroit, Michigan
WILLIAM R. EYLER, M.D.
Detroit, Michigan

The Radioisotope Renogram

Physical and physiologic factors affecting the renogram will be discussed with consideration of techniques for reducing errors. The renogram compares favorably with the other approaches to the problem of hypertension and is very valuable in determining the significance of renal artery disease.

COURSE 122

Tuesday

MORTON M. KLIGERMAN, M.D.
New Haven, Connecticut

Breast Carcinoma

An approach to the management of breast carcinoma will be developed. Consideration will be given to the choice of surgery versus irradiation, the place of steroid therapy, and the basic problem of the effect or lack of effect of treatment on survival.

The criteria of operability, the role of triple biopsy, and a presentation of recent clinical experiments will come under discussion. These latter will include (1) extended radical surgery versus the McWhirter technique, and (2) the influence of irradiation of the ovaries at the time of mastectomy.

Finally, appropriate practical treatment plans in several clinical situations will be demonstrated. A classic bibliography will be made available.

COURSE 123

Tuesday

MARVIN M. D. WILLIAMS, Ph.D.
Rochester, Minnesota

Basic Physics of Nuclear Bombs

Nuclear bombs are of two types—those which result from the fission of heavy atoms and those which result from the fusion of light atoms. Devastation attributable to these bombs is caused by radiation, heat and blast. Radiations are unique in the field of explosions; heat and blast are common to all explosions but are of tremendously greater magnitude in the explosion of nuclear bombs. Radiations occur at two different times—there are tremendous amounts of radiations liberated at the time of the explosion, and there are enormous amounts of radioisotopes, formed by the nuclear reactions, which continue to give off radiations for a long time. Large quantities of these radioisotopes may be present in the fallout deposited over large areas and at long distances from the explosion.

The basic physics, associated with fission and fusion, and the magnitude and characteristics of the radiations, heat and blast which may result from the explosion of a nuclear bomb will be discussed.

COURSE 203

Wednesday

CHARLES M. NICE, Jr., M.D.
New Orleans, Louisiana

Roentgenographic Manifestations of Collagen Diseases

Many conditions have been included under the term of collagen diseases. In general, diseases involving wide-spread changes in connective tissue or in small vessels have been called collagen diseases at one time or another.

Most writers include systemic lupus erythematosus, polyarteritis nodosa, systemic scleroderma, dermatomyositis, and rheumatic fever in the collagen disease group. Rheumatoid arthritis is considered by many to be a phase of the development of the major collagen diseases described above.

The roentgenographic findings in the above conditions will be described, the material being basically related to a study of 109 patients at the University of Minnesota Hospitals, later followed by an extension of this study at Charity Hospital in New Orleans. Combinations and sequences of findings that will help one to suspect collagen diseases will be emphasized in the presentation.

COURSE 204
and
COURSE 304

Wednesday and Thursday

HAROLD G. JACOBSON, M.D.
New York, New York

Benign Bone Tumors

Course 204. In this course the roentgen characteristics of the more frequently encountered benign bone tumors (and related entities) will be described and illustrated. These will include: (1) non-osteogenic fibroma, (2) solitary osteocartilaginous exostosis, (3) hereditary multiple osteocartilaginous exostoses, (4) solitary enchondroma, (5) enchondromatosis (Ollier's disease), (6) aneurysmal bone cyst, (7) giant cell tumor, (8) benign chondroblastoma, and several other entities such as osteoid osteoma, unicameral bone cysts and pigmented villonodular synovitis which are not necessarily benign neoplasms but are frequently included under this category.

In all instances the roentgen findings will be evaluated against the background of a discussion of the underlying pathologic changes for an adequate understanding of these roentgen abnormalities.

Malignant Bone Tumors—Primary and Secondary

Course 304. In this course the roentgen characteristics of the important malignant bone tumors—both primary and secondary—will be illustrated. These will include: (1) osteogenic sarcoma, (2) Ewing's sarcoma, (3) chondrosarcoma, (4) sarcoma with Paget's disease, (5) chordoma, (6) synovioma, (7) myelomatosis, (8) reticulum cell sarcoma, (9) Hodgkin's disease, (10) lymphosarcoma, (11) leukemia, and (12) metastatic carcinoma.

As in the case of the benign bone tumors, the roentgen findings will be illustrated against the background of a discussion of the underlying pathologic changes for an adequate understanding of these roentgen abnormalities.

COURSE 205

Wednesday

ROBERT E. WISE, M.D.
Boston, Massachusetts

Intravenous Cholangiography

Intravenous cholangiography retains its important position among the procedures of the radiologist concerned with diagnostic problems of the biliary tract. Data presented in the course have been gleaned from 8 years' experience with approximately 3,000 injections. Two thousand and thirty-two cases have been subjected to statistical analysis. Roentgenologic preoperative diagnoses have been correlated with operative findings in 466 cases, of which 225 were in the postcholecystectomy state.

Technique and interpretation will be considered in detail with emphasis on the postcholecystectomy patient as related to the time-density retention concept. Pitfalls have been analyzed and will be presented. Specific findings secondary to calculi, tumors, strictures, fibrosis of the sphincter of Oddi, pancreatitis, etc., have been analyzed for presentation.

The great value of the procedure lies in the accuracy with which organic biliary tract disease can be confirmed or excluded in the postcholecystectomy patient. Although the accurate diagnosis of a pathologic state may lead to surgical relief, at least of equal value is the exclusion of unnecessary surgical exploration.

COURSE 207
and
COURSE 307

Wednesday and Thursday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Some Fundamentals in Chest Roentgen Interpretation

Chest roentgen interpretation should not merely represent an attempt to correlate particular shadows with specific diseases, but should be approached with an understanding of the basic principles of anatomy,

physiology, pathology, and radiology involved. Some of these principles will be considered and their practical application illustrated.

Among the subjects to be discussed are:

Course 207. (1) A method of fluoroscopy; (2) the "silhouette" sign; and (3) the air bronchogram.

Course 307. (1) Anatomic variations in the pulmonary fissures; (2) lobar collapse; (3) lobar enlargement; and (4) the disrupted fissure.

COURSE 208

Wednesday

BERNARD S. EPSTEIN, M.D.
New Hyde Park, New York

Cervical Myelography and Basilar Cisternography

After a brief review of the technique of cervical myelography and the introduction of contrast material into the accessible basilar cisterns, the normal roentgen anatomy of the cervical spinal canal and basilar cisterns will be discussed. The changes incident to discogenic disease and tumors of the cervical spinal canal and meninges will be illustrated. The differential diagnosis of diseases of the spinal cord and meninges will be considered.

COURSE 221

Wednesday

J. R. CUNNINGHAM, Ph.D.
Toronto, Ontario, Canada

Problems in Clinical Dosimetry

In radiotherapy a certain dosage of radiation is specified by the therapist. The exact quantity of radiation is specified by the therapist. The exact quantity of radiation required for the eradication of the tumor is not known. Assuming however that it is, it is the duty of the physicist, the technician and the radiologist to make certain that the prescribed quantity is delivered with as much exactitude as possible.

The intent of the course is to deal with problems encountered in the carrying out of this aim. Consideration will be given to the calibration of the therapy unit and to the factors which affect the output and radiation pattern of the machine. The effect of body inhomogeneities and surface irregularities will be discussed along with various techniques that are used to compensate for this, such as bolus, transmission dosimetry, wedge fields and compensating filters. The system of on-axis dosimetry will also be discussed. Throughout the course, special emphasis will be placed on cobalt 60 and cesium 137 teletherapy.

COURSE 222

Wednesday

MELVILLE L. JACOBS, M.D.
Duarte, California

Supervoltage Radiation Therapy—Problems and Pitfalls

The instructor's experience dates back to 1935 and encompasses a study of 700 patients with advanced carcinoma of the cervix, prostate, kidney, esophagus and testicular neoplasms treated with 1,000 kv. roentgen rays at the California Institute of Technology. Since 1955, the experience includes the use of cobalt 60 and cesium 137 for the treatment of many malignant neoplasms.

The problems encountered in cobalt and cesium teletherapy are similar to those encountered in 1,000 kv. roentgen therapy. Skin sparing effects are achieved with supervoltage, but subcutaneous fibrosis is a late sequela. One must be alert to the risks involved in delivering high tumor doses through one or two fields which cross-fire, since skin dosage is much higher than tumor dose.

The relative biologic efficiency of the supervoltage should be compared with that of orthovoltage in treatment planning. The difference in absorption should be considered.

Localizations by means of films exposed with the therapy beams are safeguards. The techniques for this, employing cobalt 60 and cesium 137 beams, are to be described.

The comparative values of orthovoltage and supervoltage therapy are to be outlined.

COURSE 223

Wednesday

JACK S. KROHMER, Ph.D.
Dallas, Texas

Basic Radiobiology

I. Scope of radiobiology including radiotherapy, radiation research, radiation protection, and basic nonradiation research in which radioactive materials are used.

II. Radiation dosimetry including general concepts of dosimetry; radiation dose units including their advantages and limitations; and methods of dose evaluation and planning.

III. Initial effects of radiation on biologic material including direct or physical effects, and indirect or chemical effects; the target theory including target theory definitions, types of interactions, and important parameters to be considered in using the target theory.

IV. Time-dose relationships.

V. Radiation modifiers.

COURSE 301
and
COURSE 401

Thursday and Friday

RICHARD G. LESTER, M.D.
Richmond, Virginia

Angiocardiography in the Diagnosis of Operable Heart Disease

The place of angiocardiography and aortography in the diagnosis of many congenital cardiac lesions and certain acquired diseases is now well established. In these sessions emphasis will be placed on those conditions that are amenable to surgical correction or amelioration. Correlation with postmortem anatomy will be provided in large part by comparison of angiocardiograms and specimens of individual patients.

Introduction to Angiocardiography

Course 301. This session will begin with a discussion of indications for angiocardiography. Methods and techniques will be presented. The equipment necessary for both forward and selective examinations will be noted and the relative merits of rapid-film units and cineradiographic apparatus will be discussed.

Methods for the analysis of angiocardiographic examinations will follow. Criteria for the diagnosis of the more common lesions will be given. Data of importance to the surgeon will be emphasized.

Advanced Angiocardiography

Course 401. A more detailed analysis of certain of the findings in lesions discussed in the previous session will be provided. In addition, there will be consideration of other lesions of relative rarity but of surgical significance. The place of angiocardiography as a research method will be touched upon.

Participants are invited to show and discuss problem cases during the second session.

COURSE 302

Thursday

WILLIAM R. EYLER, M.D.
Detroit, Michigan
ROBERT S. ORMOND, M.D.
Detroit, Michigan

The Acute Abdomen

This course is designed primarily to review the roentgenologic manifestations of acute abdominal disease, particularly, as manifested on plain abdominal films. When appropriate for illustrative purposes, associated examinations with contrast materials will be presented. The following outline includes the subjects to be discussed:

1. Anatomy of the peritoneal cavity as shown by pneumoperitoneum.
2. Differentiation of the hollow viscera.
3. Vascular diseases: (a) aneurysms, dissecting and arteriosclerotic; (b) occlusive vascular disease; (c) blood dyscrasias; (d) polyserositis; (e) collagen diseases.
4. Inflammatory diseases of hollow viscera: (a) acute appendicitis, acute cholecystitis, gastritis, enteritis and cholecystitis; (b) regional enteritis; (c) tuberculosis; (d) generalized peritonitis; (e) localized abscesses.
5. Inflammatory diseases of solid viscera: (a) acute pancreatitis; (b) acute perinephric and obstructive urinary tract diseases.
6. Mechanical obstructions of the digestive tube: (a) neoplastic diseases; (b) extrinsic pressure; (c) special mechanical obstructions including intussusception, volvulus, hernia and band.
7. Traumatic conditions: (a) solid viscera; (b) hollow viscera.
8. Miscellaneous conditions causing or simulating the acute abdomen including sprue, neurologic disorders, toxic agents and endocrine malfunctions.

COURSE 303

Thursday

ROBERT P. BARDEN, M.D.
Philadelphia, Pennsylvania

The Relation of Normal and Abnormal Lung Function to Radiologic Diagnosis

Anatomic catalogues of lung diseases are without challenge to the imaginative radiologist. To understand the dynamic features of pulmonary disease, one needs information about the marvelously complex function of the lung. Some knowledge of normal and abnormal physiology makes radiologic diagnosis of lung conditions more interesting, intelligent and significant.

COURSE 305

Thursday

PHILIP RUBIN, M.D.
Rochester, New York

The Development of a Dynamic Classification of Bone Dysplasias

There is a compelling logical order to the way normal bones model. From a study of the development of normal bone growth and an analysis of the physiologic processes, one may be able to provide the key to unlock the abnormal pathophysiology in dysplastic bone diseases. An attempt is made to set up criteria for true bone dysplasias, based on an understanding of experimental and clinical pathophys-

iology. It is hoped that one can develop, on this basis, a dynamic classification for this seemingly complicated group of diseases. The differential diagnostic points of different bone dysplasias will be emphasized.

COURSE 306

Thursday

EDWARD B. SINGLETON, M.D.
Houston, Texas

Pediatric Chest Abnormalities

1. Technique
2. The normal chest and variations of normal
3. Abnormalities of the thoracic cage
4. Newborn pulmonary complications: a. variations of perinatal distress—hyalin membrane disease, aspiration syndrome and pulmonary hemorrhage; b. pneumonias including aspiration secondary to pharyngeal incoordination and esophageal atresia
5. Congenital anomalies: congenital lobar emphysema, congenital cystic malformation, diaphragmatic abnormalities, pulmonary agenesis and hypoplasia
6. Pulmonary abnormalities acquired in later infancy and childhood: a. types of pneumonia and their complications; b. acquired tracheoesophageal fistula; c. foreign bodies; d. fibrocystic disease; e. bronchiolitis
7. Rare pulmonary conditions: pulmonary alveolar microlithiasis, neoplastic diseases, pulmonary fibrosis, hemosiderosis, cytomegalic inclusion disease

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COURSE 308

Thursday

KURT AMPLATZ, M.D.
Minneapolis, Minnesota

Percutaneous Arterial Catheterization Techniques and their Applications

The presently used percutaneous catheterization technique at the University of Minnesota Hospitals will be discussed in detail and shown in motion pictures.

Different types of catheter materials, guide wires and injection equipment will be considered. The procedure of selective catheterization of branches of the abdominal aorta and, specifically, selective renal arteriography will be demonstrated on a phantom. Slides of normal and abnormal renal arteriograms with pitfalls of interpretation will be shown. Catheterization techniques of the vertebral artery via femoral artery or subclavian artery will be illustrated, the subclavian artery catheterization technique being shown in motion pictures. Aortography and left ventriculography with its indications will be presented. The technical details of selective coronary arteriography by percutaneous subclavian artery catheterization will be given. A motion picture on transthoracic percutaneous left ventricular catheterization will be shown.

Errors in technique and complications of these procedures will be stressed.

COURSE 309

Thursday

STEVEN J. FIGIEL, M.D.
Detroit, Michigan
LEO S. FIGIEL, M.D.
Detroit, Michigan

The Colon: Roentgenographic Considerations

The material presented in this course will establish evidences of a relationship between the colonic polyp and colonic cancer. The detection, significance and follow-up studies of selected case material dealing with the colonic polyp will be presented.

The incidence of error in the detection of colonic cancer will be discussed and measures to prevent such errors will be stressed.

The ileocecal region and the appendix will be covered in detail. The remainder of the course will deal with the barium enema examination in various obstructive conditions other than carcinoma, *e.g.*, volvulus, colo-colic intussusception and others. The more unusual entities in which a specific diagnosis is frequently possible such as endometriosis, pneumatosis coli, etc., will also be considered.

COURSE 310
and
COURSE 410

Thursday and Friday

JOHN A. CAMPBELL, M.D.
Indianapolis, Indiana
E. C. KLATTE, M.D.
Indianapolis, Indiana

Cineradiography

That cineradiography is a useful research method and teaching aid has been established. It has also received wide acceptance as a practical clinical method in the cardiovascular, genitourinary, and gastrointestinal fields and is gaining popularity in orthopedic, neurologic, and certain other applications. Some type of image intensification must be utilized to permit an acceptable radiation dose to the patient. This may result in a reduction in desirable image detail and limitation in field size.

The diagnostic yields obtained with currently available equipment may be enhanced by a proper understanding of the inherent limitations of the method, the controllable technical factors, and the

requirements for successful clinical adaptations. This course will present the technical and clinical considerations in cineradiography as outlined below.

Course 310. *Part I—Technical Considerations:* 1. Historical. 2. Current types of cine systems. 3. Roentgen-ray tube requirements. 4. Table adaptations. 5. Optical systems. 6. Cameras. 7. Image resolution, brightness and contrast requirements. 8. Exposure factors. 9. Kinescopic versus direct recording. 10. Film characteristics. 11. Processing. 12. Film projection and viewing. 13. Filing. 14. Radiation dose. 15. Control of image quality.

Course 410. *Part II—Clinical Considerations:* 1. Advantages and disadvantages of clinical cineradiography. 2. Clinical methodology of (a) cinecardioangiography, (b) cystourethrography, (c) orthopedic applications, (d) neuroroentgenology, (e) gastrointestinal applications, (f) gynecologic applications, (g) respiratory applications. 3. Future developments.

COURSE 321

Thursday

J. ROBERT ANDREWS, M.D.
Bethesda, Maryland

Radiobiological Basis of Radiation Therapy

The findings of cell radiobiology, as the critical system and as a model for radiotherapy, will be presented with emphasis on the following aspects:

1. The radiation response phenomena which occur following the transfer of radiation energy to the biologic system.
2. The relationship of oxygen to the radiation response (the oxygen effect).
3. The interrelationship of the oxygen effect, LET, and RBE.
4. The significance of recovery phenomena.

The implications of these aspects of cell radiobiology for human cancer radiotherapy will be discussed.

COURSE 322

Thursday

MILTON FRIEDMAN, M.D.
New York, New York

Combined Radiation and Chemotherapy in the Treatment of Human Cancer

Approximately 150 cancer patients were treated with radiation combined with chemotherapy under experimental control conditions.

The most interesting results were achieved in a series of cases with recurrent breast cancer nodules on the chest wall. In each patient, some lesions received radiation only, some 5-fluorouracil only, and some combined treatment. The observations demonstrated that combined therapy was more efficient than either modality alone, and that simultaneous administration of both modalities was more effective than consecutive therapy.

In squamous cell cancers of the head and neck, enhancement occurred less often (40 per cent of the cases) but the potential increase in cure rate was greater than in breast cancer. Methotrexate is first given by mouth or by infusion, and then followed with a sublethal dose of roentgen rays. Four consecutive cases of carcinoma of the faucial arch showed remarkable response to combined treatment. Significant responses were also seen, though less often, in cancer of the tongue, pharynx and nasal accessory sinuses.

Serial biopsy studies of the tumor added useful information.

In cancers other than the above types, enhancement effect was occasionally demonstrated.

COURSE 323

Thursday

RIEVA ROSH, M.D.
New York, New York

Clinical Management of Cancer of the Cervix Uteri

The technique of radiation therapy of cancer of the uterine cervix will be described as performed in Bellevue Hospital. A number of patients are subjected to a surgical procedure and then referred for radiation in cases of recurrent or residual cancer.

The patients can be divided into the following groups:

1. Primary—any stage—not treated by radiation or surgery prior to admission.
2. Treated with radiation, but have recurrence or residual disease.
3. Treated with surgery, but have
   a. recurrence, locally,
   b. advance of the disease found on exploration
      (I) fixation
      (II) involvement of the lymph nodes above the pelvic area, and/or the para-aortic lymph nodes.
4. Combination of all.

The plan of therapy is devised on an individual basis.

1. Cobalt 60 therapy.
2. Conventional roentgen therapy with 250 kv.
3. Radium insertion: locally, topical application in a mold or specially constructed applicator; occasionally interstitial radium is used in suitable cases.

Remission in all types of cases is obtained.

Most satisfactory results are observed in primary nontreated cases even in the more advanced stages of the disease.

COURSE 402

Friday

PAUL A. RIEMENSCHNEIDER, M.D.
Syracuse, New York

Stenosis, Occlusion and Degenerative Disease of the Cerebral Vessels

A discussion will be given considering the indications for cerebral angiography in cerebral vascular insufficiency, and the methods and hazards of such studies. Attention will be paid to the differentiation of real from spurious narrowings. Some time will be spent on the clinical and roentgenologic correlation of these abnormalities.

COURSE 403

Friday

MARTIN H. WITTENBORG, M.D.
Boston, Massachusetts

Pelvis and Hips in Infancy and Early Childhood

The roentgenologic diagnostic aspects of the common problems encountered in infancy and childhood will be discussed. These will include: synovitis, roentgenologic determination of the position of the hip, predislocation thesis, malposition, hypermobility and dislocation; delayed pubic development, achondroplasia, Hurler's and Morquio's syndrome.

COURSE 404

Friday

ROBERT H. FREIBERGER, M.D.
New York, New York

The Roentgen Examination of the Foot

The interpretation of the roentgen examination of the foot with the exceptions of fractures and dislocations is very frequently delegated to the orthopedic surgeon. The course is designed to give the radiologist a basic knowledge of tarsal and forefoot alignment.

The following points will be covered:

1. Technique of roentgen-ray examination.
2. An explanation of the terms varus and valgus and how they apply to the foot and ankle. Why the changes in tarsal alignment, primarily of the talus and calcaneus, can be expressed in terms of varus and valgus. A minimal amount of anatomy will be included.
3. The normal foot.
4. Specific foot deformities such as flatfoot, metatarsus adductus, metatarsus varus, talipes equinovarus, congenital plantar flexion of the talus, calcaneus and calcaneo-cavus.
5. Recognition of tarsal coalitions.

The course is designed to correlate physical and roentgen examination.

COURSE 405

Friday

WILLIAM E. GANNON, M.D.
Brooklyn, New York

The Localization of Intracranial Masses by Cerebral Angiography

This course will consist of a method of analyzing a cerebral angiogram to localize a space-occupying lesion. It will be assumed that there is no tumor stain present. The various types of displacement of the anterior cerebral artery, the frontopolar sign and the falx sign will be demonstrated. The significance of elevation of the middle cerebral group will be discussed. The value of the capillary and venous phases in the localization of mass lesions will be illustrated. Examples of the near and distant effects of a mass lesion will also be included.

COURSE 406

Friday

MAURICE D. SACHS, M.D.
Cleveland, Ohio

Pneumoarthrography of the Knee

Pneumoarthrography is a valuable aid in internal derangements of the knee joint. Knee joint anatomy, roentgenographic technique, normal and abnormal pneumoarthrograms will be discussed. In addition to the various types of meniscal tears, there will be many illustrations of various forms of synovitis.

COURSE 407

Friday

JULIAN O. SALIK, M.D.
Baltimore, Maryland

Early Radiographic and Cineradiographic Recognition of Pancreatic Carcinoma

The radiologic pattern of larger neoplasms of the head of the pancreas has been adequately described. However, the presence of subtle changes on the mesial border of the duodenum is often overlooked and it usually represents the first manifestations of pancreatic carcinoma. The thesis that early, clinically non-jaundiced and unsuspected changes of the pancreas can be detected radiologically will be developed, and a number of representative cases shown. In addition to slides, cineradiographic studies will be shown and their application and usefulness as compared to conventional procedures evaluated. Also, the differential diagnosis of various benign and malignant lesions of the pancreatico-ampullary area will be discussed and illustrated.

COURSE 408

Friday

GERALD D. DODD, M.D.
Philadelphia, Pennsylvania
ROY R. GREENING, M.D.
Philadelphia, Pennsylvania
SIDNEY WALLACE, M.D.
Philadelphia, Pennsylvania
HARRY W. FISCHER, M.D.
Iowa City, Iowa

Lymphangiography

This discussion will include a brief review of the historical and experimental background of lymphangiography and detail the technique, indications and complications of the intralymphatic injection of various contrast media. The roentgen appearance of normal lymphatic channels and lymph nodes will be stressed with comment on normal variants which frequently lead to erroneous interpretation. Various abnormal states will be illustrated and current and future avenues of investigation considered. Time will be reserved for discussion from the floor.

COURSE 409

Friday

WILLIAM H. SHEHADI, M.D.
Port Chester, New York

Radiologic Study of the Biliary Tract

Although some pathologic conditions involve primarily the liver and others the extrahepatic biliary tract, roentgenologically as well as clinically, the biliary tract should be considered as one unit. The anatomy, physiology and pathology of the biliary tract will be briefly reviewed, with special emphasis on a study of liver function tests and jaundice, bringing out the role they play in biliary tract visualization.

The course will encompass the technique of liver examination, liver function tests and jaundice, and a study of liver diseases which are roentgenologically significant.

Also the techniques of gallbladder and bile duct visualization, the relative merit of the oral and intravenous methods, and an evaluation of the contrast media used in biliary tract visualization will be covered. The commonly as well as the infrequently encountered diseases of the gallbladder and ducts and the so-called postcholecystectomy syndrome will be discussed from a roentgenologic point of view.

COURSE 421

Friday

WALTER T. MURPHY, M.D.
Buffalo, New York

Radiation Therapy of Head and Neck Cancer

Radiation therapy of head and neck tissues is elective for a variety of primary and metastatic malignant neoplasms. Such treatment should precede, follow or be concomitant with surgical or chemotherapeutic procedures.

The course will deal mainly with the use of roentgen rays and radium in the irradiation of primary cancers of the nasal passage, accessory nasal sinuses, oral cavity, pharynx (nasal-, oral- and laryngo-) and larynx.

Discussion will include the practical aspects of irradiation with accessible "conventional" equipment (including transoral, roentgen-ray cone techniques) as well as higher energy sources and wedge filter techniques. Complications of irradiation will be illustrated.

COURSE 422

Friday

CHAHIN M. CHAHBAZIAN, M.D.
Colorado Springs, Colorado

Radiotherapy of Carcinomas of the Skin

Many carcinomas of the skin can be treated adequately by surgery. A sizable number of others, however, is best treated by radiotherapy. Control of the lesions with ionizing rays is easily accomplished in most instances. Adequate planning and technique are prerequisite for attaining good cosmetic results which justify the choice of radiotherapy.

It is the aim of this course to outline the different technicalities and principles involved in radiotherapy to obtain the best cosmetic results in the treatment of carcinoma of the skin while maintaining the highest cure rate. Special emphasis will be placed on:

1. The choice of lesions to be treated with radiotherapy.
2. The quality of radiations to be used.
3. The special protective devices in the treatment of lesions overlying bone and cartilage and lesions of eyelids and lips.
4. The indications for proper fractionation.
5. Presentation of examples of results.

COURSE 423

Friday

JOHN G. McAFEE, M.D.
Baltimore, Maryland

The Technique and Diagnostic Applications of Scintillation Scanning

The instrumental requirements for an adequate scintillation scanning system will be briefly discussed, including methods for assessment of collimators, gamma spectrometers, and photorecording systems of data presentation.

The technique and results of scintillation scanning will be illustrated for the following clinical applications:

(1) thyroid scanning with I^{131} and I^{125} sodium iodide;
(2) brain tumor localization with I^{131} albumin and other substances;
(3) hepatic scanning with Au^{198} colloidal gold and I^{131} rose bengal;
(4) splenic scanning with sensitized Cr^{51} labeled red blood cells;
(5) renal scanning with Hg^{203} neohydrin; and
(6) mediastinal scanning with I^{131} albumin and I^{131} cholografin.

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **October 2, 1962**

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
101—Marshak	The Small Intestine	**121—Du Sault Eyler**	The Radioisotope Renogram
102—Hope	Abdominal Mass Lesions in Infants and Children	**122—Kligerman**	Breast Carcinoma
103—Morgan	Screen Intensification and Cineradiography	**123—Williams**	Basic Physics of Nuclear Bombs
104—Meszaros	Roentgen Cardiology of Adults		
105—Tuddenham	New Concepts in Radiographic and Interpretative Techniques		
106—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
107—Weens Gay	Roentgenology of the Pulmonary Circulation—Diagnosis of Certain Physiologic Changes		
108—Egan	Over Six Years' Experience with Mammography		
109—Macmillan	Sinuses		
110—Nelson	Bronchography		

CONDENSED SCHEDULE OF COURSES

WEDNESDAY 3:00 to 4:30 P.M. October 3, 1962

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
201—Marshak	The Small Intestine and Colon	221—Cunningham	Problems in Clinical Dosimetry
202—Hope	Mediastinal Mass Lesions in Infants and Children	222—Jacobs	Supervoltage Radiation Therapy—Problems and Pitfalls
203—Nice	Roentgenographic Manifestations of Collagen Diseases	223—Krohmer	Basic Radiobiology
204—Jacobson	Benign Bone Tumors		
205—Wise	Intravenous Cholangiography		
206—Etter	Normal and Pathologic Roentgen Anatomy of the Skull and Paranasal Sinuses		
207—Felson	Some Fundamentals in Chest Roentgen Interpretation		
208—Epstein	Cervical Myelography and Basilar Cisternography		
209—Macmillan	Mastoids, Lateral Neck and Larynx		
210—Nelson	Bronchography		

CONDENSED SCHEDULE OF COURSES

THURSDAY 3:00 to 4:30 P.M. October 4, 1962

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
301—Lester	Introduction to Angiocardiography	321—Andrews	Radiobiological Basis of Radiation Therapy
302—Eyler Ormond	The Acute Abdomen	322—Friedman	Combined Radiation and Chemotherapy in the Treatment of Human Cancer
303—Barden	The Relation of Normal and Abnormal Lung Function to Radiologic Diagnosis	323—Rosh	Clinical Management of Cancer of the Cervix Uteri
304—Jacobson	Malignant Bone Tumors—Primary and Secondary		
305—Rubin	The Development of a Dynamic Classification of Bone Dysplasias		
306—Singleton	Pediatric Chest Abnormalities		
307—Felson	Some Fundamentals in Chest Roentgen Interpretation		
308—Amplatz	Percutaneous Arterial Catheterization Techniques and their Applications		
309—Figiel Figiel	The Colon: Roentgenographic Considerations		
310—Campbell Klatte	Cineradiography. Part I. Technical Considerations		

CONDENSED SCHEDULE OF COURSES

FRIDAY 1:30 to 3:00 P.M. October 5, 1962

Diagnostic Roentgenology		Therapeutic Radiology, Nuclear Medicine, Physics and Radiobiology	
401—Lester	Advanced Angiocardiography	421—Murphy	Radiation Therapy of Head and Neck Cancer
402—Riemenschneider	Stenosis, Occlusion and Degenerative Disease of the Cerebral Vessels	422—Chahbazian	Radiotherapy of Carcinomas of the Skin
403—Wittenborg	Pelvis and Hips in Infancy and Early Childhood	423—McAfee	The Technique and Diagnostic Applications of Scintillation Scanning
404—Freiberger	The Roentgen Examination of the Foot		
405—Gannon	The Localization of Intracranial Masses by Cerebral Angiography		
406—Sachs	Pneumoarthrography of the Knee		
407—Salik	Early Radiographic and Cineradiographic Recognition of Pancreatic Carcinoma		
408—Dodd Greening Wallace Fischer	Lymphangiography		
409—Shehadi	Radiologic Study of the Biliary Tract		
410—Campbell Klatte	Cineradiography. Part II. Clinical Considerations		

NEWS ITEMS

DR. FAY H. SQUIRE HONORED

Dr. Fay H. Squire, Professor of Radiology at the University of Illinois College of Medicine, and Chairman of the Radiology and Nuclear Medicine Division, Presbyterian-St. Luke's Hospital, Chicago, was honored recently at a dinner in the Palmer House by the men he had trained in radiology during 32 years.

Fifty doctors and their wives, representing practicing radiologists from Maine to California, watched as a portrait of Dr. Squire was presented to him. The portrait, by Chicago artist Gustaf Dalstrom, has been hung in the radiology waiting room.

POSTGRADUATE COURSE
RADIOLOGY OF THE GASTRO-INTESTINAL TRACT
EMORY UNIVERSITY

The Department of Radiology of Emory University School of Medicine will conduct a postgraduate course, Radiology of the Gastrointestinal Tract, on October 19 and 20, 1962. This course will be held at Grady Memorial Hospital, Atlanta, Georgia.

The guest faculty will be Drs. Richard H. Marshak, New York, N. Y.; Richard Schatzki, Boston, Mass.; and Edward B. Singleton, Houston, Texas.

For further information, please write to H. S. Weens, M.D., Department of Radiology, Emory University School of Medicine, Atlanta 22, Georgia.

COURSES AT THE QUEENS HOSPITAL CENTER

The following courses will be offered at the Queens Hospital Center, Department of Hospitals, The City of New York, in the Fall of 1962:

(1) *Course in Radioactive Isotopes for Technicians*, consisting of 8 sessions of 4 hours each, to be held every Wednesday afternoon, commencing September 26, 1962 and ending November 14, 1962 (1 to 5 P.M.). Tuition: $100.00.

(2) *Operative Radium Therapy Day*, to be held October 17, 1962. Registration: $10.00.

(3) *Special Radiation Procedures in the Treatment of Cancer;* this is a one week intensive course in the use of specialized radiation techniques in the treatment of malignant disease commencing October 15, 1962. Tuition: $150.00.

For further particulars, please write to Dr. Philip Kahan, Supervising Medical Superintendent, Queens Hospital Center, 82–68 164th Street, Jamaica 32, New York.

SYMPOSIUM ON CLINICAL RADIOISOTOPE SCANNING

A five day symposium on clinical radioisotope scanning will be held at the Medical Division, Oak Ridge Institute of Nuclear Studies, and supported by Division of Biology and Medicine, U. S. Atomic Energy Commission, October 22–26, 1962.

Experts from throughout the country will lecture, demonstrate equipment and exhibit scans. The topics will include: scanning and recording systems; approaches to thyroid, brain, liver, kidney, spleen scanning; interpretative techniques by instrumentation; evaluation of present scanning systems; daily workshops on interesting and problem scans from faculty and participants.

The symposium is planned for physicians and paramedical personnel with previous radioisotope experience. The maximum enrollment is 40.

For enrollment please write to Dr. Ralph M. Kniseley, Medical Division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tennessee.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

GLOSSARY OF WORDS AND PHRASES USED IN RADIOLOGY AND NUCLEAR MEDICINE. By Lewis E. Etter, B.S., M.D., F.A.C.R., Professor of Radiology and Chief, Radiological Service, Western Psychiatric Institute and Falk Clinic, School of Medicine, University of Pittsburgh. Cloth. Price $8.50. Pp. 203. Charles C Thomas, Springfield, Illinois.

This book, titled a "Glossary," is more than that since it also contains sections on suggested terminology for roentgenologic reporting and an excellent discussion of radiologic symbols and semantics. The addition of new descriptive terms in any scientific field necessitates their collection under one roof. Emphasis on old words by dissemination of their meaning and spelling for medical secretaries, editors and others is a valuable contribution to radiologic literature. This book is comprehensive and complete in detail, but is concise and compact. Words and phrases are alphabetically arranged, permitting quick reference. Its scope is unusually wide, ranging from terms intended for the technican such as "pass box," to rare terms of interest to the nuclear physicist and radiologist such as "spallation." The transuranium elements and even major American radiologic organizations and publications are listed. The conventional medical dictionaries are not specifically oriented to this specialty and are not always accurate nor complete in the definition of its terms. Of particular use to secretaries is the clarification of certain homonyms, the most interesting of which Dr. Etter cites as being the foraminal valley and bazaar, bizarre, and bezoar.

In discussing radiologic symbols and semantics, the terms "x-ray," "plate," "intravenous pyelogram," "Bucky," and "portable" are questioned as to correctness and a brief etymologic description of their origin is presented.

Dr. Etter bodes well to be the Webster of radiology. An x-ray surgical appliance chart is included, although it is of necessity incomplete due to the great number of such devices.

A section on suggested terminology for roentgenologic reports, conveniently indexed and outlined, was taken from material prepared for the educational committee of the Pennsylvania Radiological Society by the Standard Report Form Committee with some additions and revisions. A further aid in accurate reporting presents 21 sample roentgenologic reports, paradigms of reporting.

Although eponyms add color to our medical language and frequently serve as shorthand expressions, those which allude to quite different pathologic processes may properly be eliminated, *viz.*, "Paget's disease,"—an affection of bone or the breast. Similarly, "lower esophageal ring" is preferred to "Schatzki's ring" and the "inverted 3 sign" is more acceptable than "Frostberg's epsilon sign."

The latest revision of the Basle Nomina Anatomica compiled by the International Congress of Anatomists in Paris, 1955, should be studied by all radiologists. The new Nomina Anatomica, however, does not contain new usages such as "pars interarticularis" or "outflow tract" of the heart.

This book reflects the scholarship, organizational ability and etymologic interests of its distinguished author and will prove an invaluable aid to secretaries, residents and others but may also be read with interest and profit by radiologists.

K. L. KRABBENHOFT, M.D.

AN ATLAS OF ESOPHAGEAL MOTILITY IN HEALTH AND DISEASE. By Charles F. Code, M.D., Ph.D., Professor of Physiology, Mayo Foundation; Consultant, Section of Physiology, Mayo Clinic; Brian Creamer, M.D., M.R.C.P. (London), Research Assistant, Section of Physiology, Mayo Clinic and Mayo Foundation; Jerry F. Schlegel, B.S., Technical Assistant, Section of Physiology, Mayo Clinic; Arthur M. Olsen, M.D., M.S. in Medicine, Associate Professor of Medicine, Mayo Foundation; Consultant, Section of Medicine, Mayo Clinic; F. Edmund Donoghue, M.D., M.S. in Medicine, Instructor in Medicine, Mayo Foundation; Consultant, Section of Medicine, Mayo Clinic; and Howard A. Andersen, M.D., M.S. in Medi-

cine, Instructor in Medicine, Mayo Foundation; Consultant, Section of Medicine, Mayo Clinic, Rochester, Minnesota. Cloth. Price, $8.50. Pp. 134, with many illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence, Springfield, Ill., 1960.

This is a unique presentation of a series of charts and graphs summarizing seven years of research on esophageal motility and pressure gradients at the Mayo Clinic. The associated explanatory text is simple and provides continuity between illustrations. The book can easily be perused in a single evening, and yet it may be valuable as a reference volume for problems in specific esophageal disease.

The motility studies deal with the normal esophagus and its sphincters as well as the conditions of achalasia, diffuse spasm, and scleroderma. It is somewhat surprising to find that higher than average pressures are common to all three of these pathologic conditions. Although there is no utilization of barium contrast studies, the radiologist cannot help but mentally transpose the illustrative tracings into his more familiar fluoroscopic impressions.

The book is highly recommended for those who would have a basic understanding of the physiologic conditions necessary for commonly observed phenomena related to the esophagus.

Arch H. Hall, M.D.

Clinical Radiology of the Oesophagus. By Marcel Brombart, Director of the Department of Radio-diagnosis at the César de Paepe Clinic, Brussels. Translated by Sheila Kenny, M.A., M.D. (Dubl.), Consultant Radiologist, Armagh City Hospital. Cloth. Price, $15.00. Pp. 383 with numerous illustrations. Publisher, The Williams & Wilkins Company, Baltimore 2, Md., exclusive United States agents, 1961.

In his finely written book Dr. Brombart has accomplished an excellent task in furthering the knowledge concerning the esophagus and its function. He has culled from the numerous articles about the esophagus the many theories regarding its more complicated functions and abnormalities and has tried to substantiate or disprove these from his own vast clinical experience.

He has presented what he considers the essentials of a good fluoroscopic and film study of the esophagus and detailed his own method. A brief review of the anatomy and physiology of the esophagus has been included. A thorough discussion of the functional disorders of the pharynx, thoracic esophagus and of the cardiophrenic segment of the esophagus adds much to the readers knowledge concerning these important aspects of the esophagus.

He has provided us with a complete and orderly analysis of the effects upon the esophagus from extrinsic factors. Beginning with the results from cervical disks, he discusses the tumors, adhesions and congenital vascular impressions at the various levels. Of particular interest is the relationship of the esophagus to the aorta as it unfolds and elongates and of the special incidence where it can present in both the left and right anterolateral positions during the same examination.

The section of the book devoted to diverticula of the esophagus explodes the previously held views regarding statistical occurrence of pharyngoesophageal diverticula. The author states that diverticulum of the thoracic esophagus is ten times more common than that of the pharyngo-esophagus. A comprehensive review of the theories regarding diverticula formation at the various levels and their appearances is given.

Esophagitis and ulceration are discussed, the causes indicated and the differential diagnoses enumerated.

The diagnosis of carcinoma of the esophagus, particularly early in the hypopharynx, is difficult. The author presents his criteria for investigation and stresses the importance of careful fluoroscopy and film timing. The roentgen appearances of the infiltrating, papilliferous and ulcerating types of carcinoma of the esophagus are shown. The signs on fluoroscopy of carcinoma in the juxta cardia are listed.

Two sections are devoted to discussion of esophageal varices and the problem of gastric herniation through the esophageal hiatus.

The most impressive fact one gains from reading this book is a realization of the minute care with which the author works. The esophagus is rapidly passed over in the routine upper gastrointestinal tract examination by many of us, yet it may be the organ of symptoms in many cases. The application of the method described by Marcel Brombart would undoubtedly lead to a higher percentage of positive findings.

The book contains 337 figures and illustra-

tions in a text of 383 pages providing the reader with ample pictorial evidence of the points discussed. It is truly a fine contribution reflecting the vast knowledge and experience of a very dedicated man.

G. A. Kling, M.D.

BOOKS RECEIVED

Annual Review of Medicine. Vol. 13. Edited by David A. Rytand, Stanford University School of Medicine; and William P. Creger, Stanford University School of Medicine. Cloth. Price, $7.00. Pp. 552. Annual Reviews, Inc., 231 Grant Avenue, Palo Alto, Calif., 1962.

Tritium in the Physical and Biological Sciences. Vol. I. Proceedings of the Symposium sponsored by the International Atomic Energy Agency in co-operation with the Joint Commission on Applied Radioactivity and held in Vienna, May 3–10, 1961. Cloth. Price, $7.00. Pp. 369, with many figures and tables. International Publications, Inc., 801 Third Avenue, New York 22, N. Y., 1962.

Tritium in the Physical and Biological Sciences. Vol. II. Proceedings of the Symposium sponsored by the International Atomic Energy Agency in co-operation with the Joint Commission on Applied Radioactivity and held in Vienna, May 3–10, 1961. Cloth. Price, $8.00. Pp. 438, with many figures and tables. International Publications, Inc., 801 Third Avenue, New York 22, N. Y. 1962.

Die Wirbelsäulenleiden und ihre Differentialdiagnose. By Prof. Dr. J. E. W. Brocher, Genf. Cloth. Pp. 537, with 342, illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart N, Germany, 1962. In the U. S. A. and Canada: Intercontinental Medical Book Corporation, New York 16, N. Y.

Whole-Body Counting. Proceedings of the Symposium held by the International Atomic Energy Agency at the Neue Hofburg, Vienna, June 12–16, 1961. Cloth. Price, $10.00. Pp. 535, with many figures. International Publications, Inc., 801 Third Avenue, New York 22, N. Y., 1962.

Radioisotopes and Radiation in Entomology. Proceedings of the Symposium on Radioisotopes and Radiation in Entomology sponsored by the International Atomic Energy Agency and held in Bombay, December 5–9, 1960. Cloth. Price, $6.50. Pp. 307. International Publications, Inc., 801 Third Avenue, New York 22, N. Y., 1962.

Le Césium 137 en Téléthérapie. By R. Amalric and J. P. Vigne. Paper. Pp. 198, with 61 illustrations. Gauthier-Villars & Cie, Éditeur, 55 Quai des Grands-Augustins, Paris 6e, France, 1962.

Atlas of Tumor Pathology. Section II—Fasc. 4. Tumors of Bone and Cartilage. By Lauren V. Ackerman, M.D., Professor of Surgical Pathology and Pathology, Washington University School of Medicine, St. Louis, Mo.; Surgical Pathologist, Barnes Hospital and Affiliated Hospitals, St. Louis, Mo.; Consultant to the Armed Forces Institute of Pathology, Washington, D. C.; and Harlan J. Spjut, M.D., Associate Professor of Surgical Pathology, Washington University School of Medicine, St. Louis, Mo.; Associate Surgical Pathologist, Barnes Hospital and Affiliated Hospitals, St. Louis, Mo. Paper. Price, $3.00. Pp. 347, with 362 illustrations. American Registry of Pathology, Armed Forces Institute of Pathology, Washington 25, D. C., 1962.

Leber und Nachbarorgane. By Dr. Leo Wannagat. Paper. Pp. 144, with many illustrations. Georg Thieme Verlag, Herdweg 63, Stuttgart N, Germany, 1962. In the U.S.A. and Canada: Intercontinental Medical Book Corporation, New York 16, N. Y.

Index Stratigraphicus (Raccolta di voci bibliografiche sulla Stratigrafia dal 1930 al 1959). By Dr. G. L. Besio. Paper. Pp. 275. Edizioni Universitarie, Corso Gastaldi 39/1, Genova, Italy, 1962.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Head

Buraggi, G. L., and Capelli, L. Osservazioni sulla stratigrafia del seno mascellare in condizioni patologiche. (Observations on laminagraphy of the maxillary sinus in pathologic conditions.) *Radiol. med.*, Dec., 1961, *47*, 1163–1181. (Address: Via Machiavelli 40, Milano, Italy.)

The authors in previous articles have discussed in detail the most common causes of error in evaluating the roentgen picture obtained by laminagraphy of maxillary sinuses in normal conditions. In this study laminagraphy is used to demonstrate pathologic changes of the maxillary sinuses and adjacent areas.

The roentgen findings are characterized by: a) changes in the cavity itself, and b) changes in the walls of the sinuses.

The necessary condition to demonstrate roentgenologically a pathologic process in the sinus is the absence of air in the sinus itself. This hyporadiolucency is of course demonstrated also by standard roentgenograms but it is only with laminagraphy that more definite information is obtained. Demonstrated are the degree of the hyporadiolucency, its extension, type, exact location, and the characters of its limits.

The principal obstacle to correct interpretation of a laminagram of the paranasal sinuses is related to the limitation of the unidirectional type of laminagraphy and in particular to the impossibility of eliminating completely the anatomic components outside the sinuses. The most simple method of reducing these sources of error to a minimum consists in performing laminagraphic studies of the maxillary sinus adopting a definite and regular technique in order to obtain a constantly similar representation of the morphology and site of the so-called transport shadows.

The changes in the wall of the sinuses are of three types: 1) osteoporosis or atrophy, 2) destruction or localized osteolysis, and 3) alteration of one of the walls.

A laminagraphic evaluation of the bony changes is always more difficult. The limitations are related to the method itself, in the sense that an osteolysis of one of the walls must be at least 1 $cm.^2$ in order to be demonstrable roentgenographically. Secondarily the limits between decalcification and destruction are very often not very definite, since at times the destruction takes place almost immediately, while in other cases the osteolysis is the end result of a slow process of reabsorption of bone.

From these studies the authors conclude that laminagraphy is important in the evaluation of pathologic changes in the paranasal sinuses and is in many cases the only procedure which permits the correct demonstration of both the nature and extent of the pathologic process.

Several demonstrative reproductions of laminagrams of the paranasal sinuses in various pathologic conditions are presented and add much to the understanding of the technical points discussed by the authors.—*A. F. Govoni, M.D.*

Dong, Nguyen Tran, Léger, Jean-Louis, and Belanger, Raymond. La signification d'un niveau liquide dans le sinus sphénoidal après un traumatisme crânien. (The significance of a fluid level in the sphenoid sinus after cranial trauma.) *J. Canad. A. Radiologists*, Dec., 1961, *12*, 113–116. (From: Département de Radiologie, Hôpital Notre-Dame, Montréal, Québec.)

Thirty-three cases of closed head injuries showed an air-fluid level in the sphenoid sinus on roentgenologic examination performed during the first few hours after trauma. The so-called "brow-up lateral" is the view used as a basic projection in the preliminary examination of head injuries. No fracture of the sphenoid sinuses was directly observed in these cases, but a detailed investigation revealed 30 cases with definite signs of injury to the base of the skull.

Although some authors have reported fluid in the sphenoid sinus in few cases with traumatic cerebrospinal fluid rhinorrhea, the present authors would like to extend the clinical significance of this extravasated fluid to include fractures of the base of the skull. In basal fractures, as well as in less severe types of head injuries, transient and unrecognized cerebrospinal fluid leakage and/or sinusal hemorrhage at the floor of the anterior fossa, may drain by gravity into the sphenoid sinuses because of their anatomic relationships. The air-fluid level sign by itself did not modify the prognosis.—*Charles M. Nice, Jr., M.D., Ph.D.*

Huber, P. Die Verletzungen der Meningealgefäße beim Epiduralhämatom im Angiogramm. (Injuries of the meningeal vessels in epidural hematomas demonstrated by angiography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1962, *96*, 207–220. (Address: Chirurgische Universitätsklinik, Inselspital, Bern, Switzerland.)

Early angiography via the external carotid artery in cases of brain and/or skull injury not only can demonstrate position and extent of the intracranial hematoma, but may quite often also show the actual point of trauma to the artery which causes the hematoma. The point of exit of contrast material from the artery into the hematoma can be visualized. The extravasation is seen in different shapes from a localized spot to more diffuse streaks.

A series of angiographic reproductions illustrates

the author's contention. Of 24 cases, there was evidence of localized extravasation in 7 cases, with questionable findings in 3. It would appear that the smaller the interval between trauma and angiography, the better the possibility to demonstrate the break in the vessel.

The author suggests to inject the contrast material into the common carotid artery in order to possibly demonstrate a direct trauma to the meningeal vessels when arteriography is indicated in skull and brain injury.—*Hans W. Hefke, M.D.*

Caprotti, M. Su un caso di tumore retrooculare diagnosticato con orbitografia. (A case of retro-ocular tumor diagnosed by orbitography.) *Ann. radiol. diag.*, 1961, *34*, 403–410. (From: Università degli Studi di Pavia, Istituto di Radiologia e Terapia Fisica, Pavia, Italy.)

The first part of this article discusses the technique of orbitography with use of either a gaseous or liquid contrast medium. The author employed a liquid contrast medium to demonstrate the normal distribution of the injected material into the retro-orbital space.

Roentgenograms were taken in frontal, lateral, and axial projections. These were also supplemented by laminagrams in the 3 mentioned projections. Best results were obtained in space occupying lesions such as tumors and cysts.

The case presented in this article is one of a teleangiectatic glioma of the optic nerve. The illustrations are convincing and not too difficult of interpretation. Accurate localization and estimation of the size of the tumor were possible in this case by orbitography.—*Peter E. Russo, M.D.*

Paillas, J. E., Bonnal, J., Legre, J., and Combalbert, A. Des méningiomes multiples à la méningiomatose en plaques au cours de la maladie de Recklinghausen. (From multiple meningiomas to meningiomatosis *en plaque* in the course of Recklinghausen's disease.) *Presse méd.*, Dec., 1961, *56*, 2604–2606. (From: Clinique Neuro-Chirurgicale de l'Hôpital de La Timone, Faculté de Médecine de Marseille, Marseille, France.)

In the course of Recklinghausen's neurofibromatosis, multiple intracranial meningiomas may develop, accompanying or following the development of neurinomas implanted on the peripheral nerves.

Three demonstrative cases are reported: meningioma *en plaque* and subarachnoid meningiomas of the parietal region in the first case; massive parasagittal and adjacent nodular meningiomas in the second case; and massive rolandic meningioma associated with meningiomatosis *en plaque* in the third case. All of the patients had in addition multiple neurofibromatous localizations on the acoustic and peripheral nerves.

Roentgenologically the findings are quite characteristic: disseminated plaques of meningeal calcification associated with opaque areas in the cranial vault.

Such cases raise the question of relationship of multiple meningiomas to meningiomatosis. It is possible that the first merges into the latter.—*Charles M. Nice, Jr., M.D., Ph.D.*

Kahana, Lawrence, Lebovitz, Harold, Lusk, Walter, McPherson, Harry T., Davidson, Eugene T., Oppenheimer, Jack H., Engel, Frank L., Woodhall, Barnes, and Odom, Guy. Endocrine manifestations of intracranial extrasellar lesions. *J. Clin. Endocrinol. & Metabol.*, Mar., 1962, *22*, 304–324. (From: Division of Endocrinology, Department of Medicine and Division of Neurosurgery, Department of Surgery, Duke University Medical Center, Durham, N. C.)

Fourteen patients with intracranial extrasellar lesions simulating primary pituitary disease are reported. Two had aneurysms of the internal carotid artery, 4 had tumors involving the third ventricle, 4 had internal hydrocephalus due to a block in the aqueduct of Sylvius, and 1 each had glioma of the optic chiasm, arteriovenous anomaly of the left cerebrum, meningioma of the floor of the anterior fossa and mucocele of the sphenoid sinus.

Thirteen patients had clinical and laboratory evidences of hypothalamic-hypophysial dysfunction. Abnormality of gonadotropin secretion occurred in 10, of TSH secretion in 6, of ACTH secretion in 5, and 4 had diabetes insipidus. All the patients with third-ventricle tumors, glioma of the optic chiasm and arteriovenous anomaly and one of those with aneurysm of the internal carotid artery had the most extensive endocrine deficiencies. In several cases, substitution therapy with cortisone was crucial in carrying the patients through stressful experiences, and in their rehabilitation. The importance of adequate endocrine substitution therapy in the management of these patients is emphasized.

In the 4 patients with internal hydrocephalus, the onset of the illness had been in childhood; they were characterized by delayed pubescence in 3 and precocious puberty with delayed menarche in the fourth. The patients with aneurysm of the internal carotid artery, internal hydrocephalus, meningioma and mucocele of the sphenoid sinus demonstrated roentgenographic changes in the skull suggesting pituitary tumor, whereas the other patients were characterized by prominent endocrine manifestations without significant alterations in the sella turcica.

Six charts and tables and ten reproductions of roentgenograms accompany this article.—*F. B. Markunas, M.D.*

Friedmann, G., Krenkel, W., and Tönnis, W. Angiographie oder Pneumographie? Vergleichende Untersuchungen bei 670 supratentoriellen Tumoren. (Angiography or pneumography? Comparative study in 670 supratentorial tumors.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1962, *96*, 181–200. (Address: Neurochirurgische Universitätsklinik, Köln, Germany.)

This study concludes that the greater percentage of supratentorial tumors can be diagnosed and localized by serial carotid angiography. Of 670 patients, cerebral angiography was done in 639. In 82 per cent of these an exact localization of the tumor was possible.

The number of tumors found and localized by skull films alone was only 0.7 per cent. In 17.5 per cent of all cases, both angiography and pneumography were necessary. The necessity of adding pneumography to angiography was dependent on the situation of the tumor. Tumors in the parasagittal, parietal and frontal regions often required pneumography for diagnosis and localization. Tumors in the neighborhood of the larger cerebral vessels can most often be diagnosed reliably by angiography alone.

It must be remembered that neither by angiography nor by pneumography is one able to diagnose all tumors in their early stage. It is at times necessary to repeat these examinations after an interval.

In every sixth case of this study, pneumography was essential to accurately diagnose or localize the tumor. In meningiomas, oligodendroglioma and glioblastoma, a pneumogram was necessary in only 10 per cent, mainly because of the "staining."—*Hans W. Hefke, M.D.*

Fuenmayor, P., and Llovet, J. Hemorragia subaracnoidea como manifestación inicial de los tumores cerebrales. (Subarachnoid hemorrhage as the initial manifestation of brain tumors.) *An. med.*, Sept., 1961, *47*, 271–279.

This paper describes 2 patients with glioblastoma multiforme who had a subarachnoid hemorrhage as the first manifestation of their disease. In one patient the correct diagnosis was made only post mortem; the other patient had an early angiography that demonstrated a brain tumor.—*F. Comas, M.D.*

Wiedenmann, O. Eine extrakranielle Anastomose zwischen dem Versorgungsgebiet der A. carotis externa und der A. vertebralis. (An extracranial anastomosis between the external carotid artery and the vertebral artery.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1962, *96*, 201–206. (Address: Neurologische Universitätsklinik, Luitpold-Krankenhaus, Würzburg, Germany.)

This is a very short report of demonstration of a small communicating artery between the ascending pharyngeal artery (a branch of the external carotid artery) and the vertebral artery by means of cerebral arteriography. The possible clinical importance under certain circumstances is discussed.—*Hans W. Hefke, M.D.*

Neck and Chest

Kamberg, Samuel, Loitman, Bernard S., and Holtz, Sumner. Amyloidosis of the tracheobronchial tree. *New England J. Med.*, Mar., 1962, *266*, 587–591. (From: Department of Radiology, Veterans Administration Hospital, the Mallinckrodt Institute of Radiology, Washington University School of Medicine, and the Department of Radiology, Jewish Hospital of St. Louis, Mo.)

Two cases of tumor-forming amyloidosis of the tracheobronchial tree are reported, thus bringing the total number in the reviewed literature to 20. These cases were diagnosed during life by bronchography and bronchoscopic biopsy.

The signs and symptoms are dependent on the size and location of the amyloid tumors, and may be hoarseness, dyspnea, cough, wheezing, hemoptysis, pneumonia, emphysema or atelectasis.

The amyloid substance is deposited in subepithelial tissues of the tracheobronchial tree, forming ridges, plaques and nodules, which indent the tracheal column and form wax-like imprints. It is improbable that a diagnosis of tracheobronchial amyloidosis will be made on conventional roentgenologic study of the chest.—*F. B. Markunas, M.D.*

Miller, Winston R., and Jay, Alan R. Staphylococcal pneumonia in influenza: five cases. *A.M.A. Arch. Int. Med.*, Mar., 1962, *109*, 276–286. (Address: 521 Hill Street, Red Wing, Minn.)

Concomitant influenzal-staphylococcal pneumonia is a dread and fearful disease with high mortality rate. Five cases of symbiotic pneumonia with influenza A virus and *Staphylococcus aureus* are presented. Three of the 5 patients died. One was given prophylactic-type treatment with usual dosage of an antibiotic to which the staphylococcus was shown to be sensitive *in vitro*, but which seemed to have absolutely no effect *in vivo*. Another died from toxemia so profound that it mimicked paralytic brain hemorrhage. The third case, in which the outcome was fatal, had a paucity of pulmonary and roentgen ray findings but prominent abdominal symptoms.

The influenza virus alone produces ulcerating bronchiolitis and massive pulmonary edema. The greatest barrier to staphylococcic infection is the intact mucous membrane. When this membrane is ulcerated by influenza virus infection, the staphylo-

coccus runs rampant. Rapid septic thrombosis of adjacent lobular arteries produces multiple septic emboli and lobular infarction. Edema, hemorrhage and bronchopneumonia cause further obliteration of alveolar spaces. Cyanosis is severe and generalized toxemia is profound.

Prospects for successful treatment of the fulminating form of this disease seem utterly hopeless. The only solution is prevention of the influenza virus infection by vaccination.—*Francis P. Shea, M.D.*

Field, C. Elaine. Bronchiectasis: a long-term follow-up of medical and surgical cases from childhood. *Arch. Dis. Childhood*, Dec., 1961, *36*, 587–603. (Address: The Paediatric Unit, Queen Mary Hospital, Hong Kong.)

A survey of 225 patients with bronchiectasis observed over a period of 8 to 21 years was conducted at the Queen Mary Hospital, Hong Kong. This is a continuation of an earlier study on 202 patients published by the author in 1949. The present series is comprised of patients between 10 and 29 years, principally between 14 and 24 years. In 169 patients, the onset was in the first five years of life; thereafter, there was some improvement, most marked around puberty, and the disease seemed to become stationary in the twenties. The results were slightly better in the surgically treated patients (121 were treated surgically and 104 medically), but the two groups were not comparable.

The effect of the use of chemotherapy is difficult to assess statistically, but it has doubtless contributed to more complete and prompt recovery from recurrent respiratory infections and therefore is significant in slowing the rate of deterioration. None of this series was treated with continuous chemotherapy.

Williams and O'Reilly (1959) described two types of bronchiectasis: (a) that with subacute pyogenic pulmonary collapse and (b) that with nonspecific infective bronchiolitis and/or interstitial pneumonia. Many cases of type (a) will show complete or partial resolution with medical management, and it is therefore important to recognize these cases which may still be considered reversible in order to avoid unnecessary surgery.

Although occasionally bronchiectasis will develop in dependent bronchi following surgical treatment for bronchiectasis, or in lung adjacent to severe bronchiectatic lung with secretions, on the whole bronchiectasis is not a spreading disease.

Growth records for height and weight tended to fall under the 50 percentile in this series, particularly in the medically treated patients.

Twelve deaths were recorded, two of which were not due to the bronchiectasis. All other fatal cases were suffering from extensive bilateral disease.—*Lois Cowan Collins, M.D.*

Gremmel, H. Die Transversalschichtuntersuchung des Herzens und der grossen Gefässe. (Transverse laminagraphy of the heart and the great vessels.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1962, *96*, 3–36. (Address: Dozent Dr. med. H. Gremmel, Moorenstr. 5, Düsseldorf, Germany.)

Transverse laminagraphy of the chest organs gives valuable information on the mediastinal structures in the third dimension. Although being an easily performed supplementary method, it is used very little. Nevertheless, for the planning of radiotherapeutic and surgical procedures it may prove important.

The author has employed the method in 181 cases in addition to regular laminagraphy, angiocardiography and kymography. The angle of the tube was 25°, the voltage 50 to 100 kv., and the exposure time 2 seconds. For the diagnosis of cardiovascular disease the following 5 planes were selected: (1) level of the aortic arch, (2) level of the pulmonary artery segment, (3) transhilar level, (4) subhilar level, and (5) a level slightly above the diaphragm.

Numerous examples of congenital and acquired cardiovascular disorders are discussed and illustrated. A tabulation shows that the plane at the diaphragmatic domes is most revealing. The only exceptions are aortic isthmus stenosis and aortic aneurysm. Rotation of the heart in either direction due to enlargement of a single chamber is easily recognized. For proper interpretation of the transverse laminagrams, however, comparison with a normal standard is helpful.—*Ernest Kraft, M.D.*





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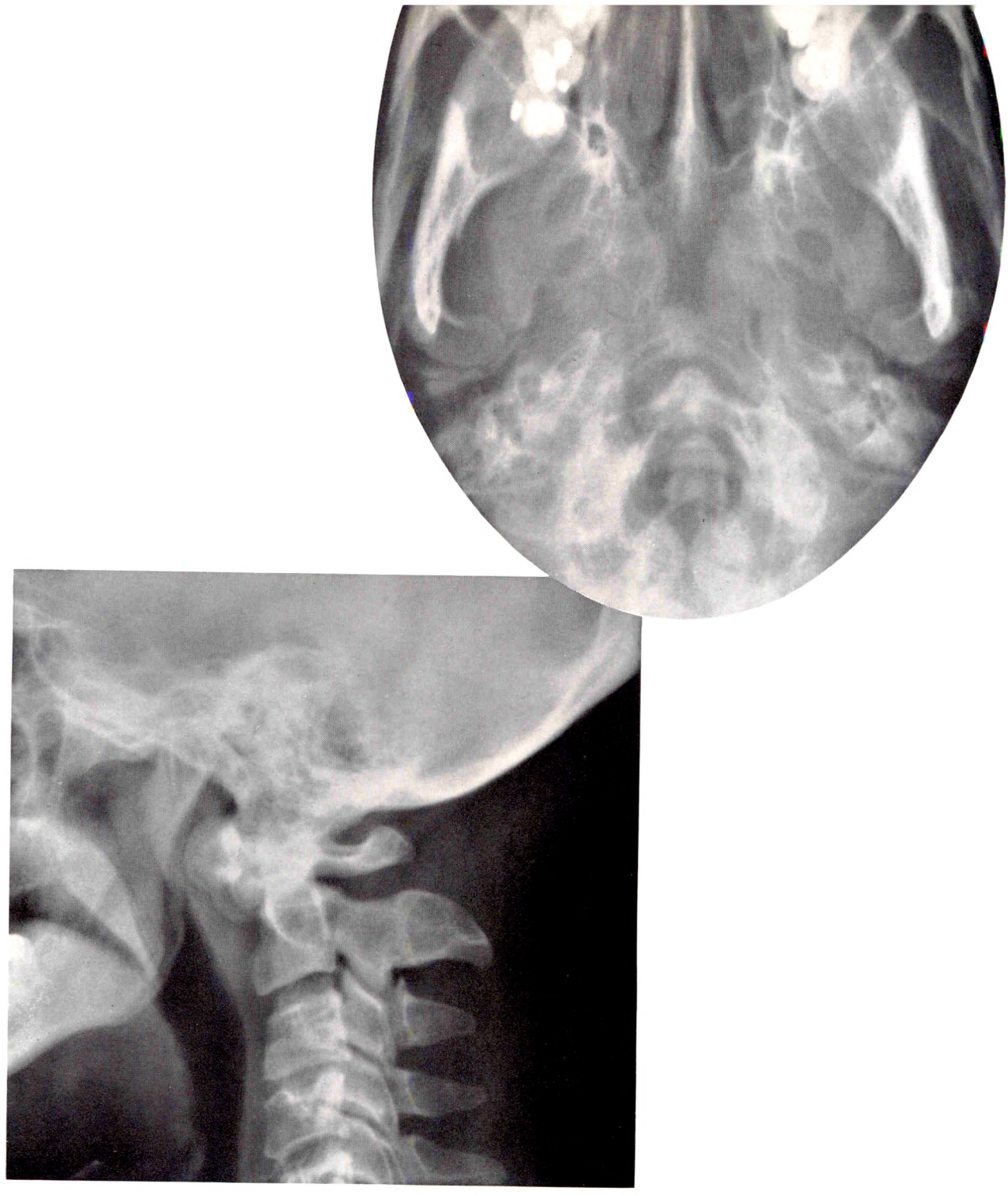


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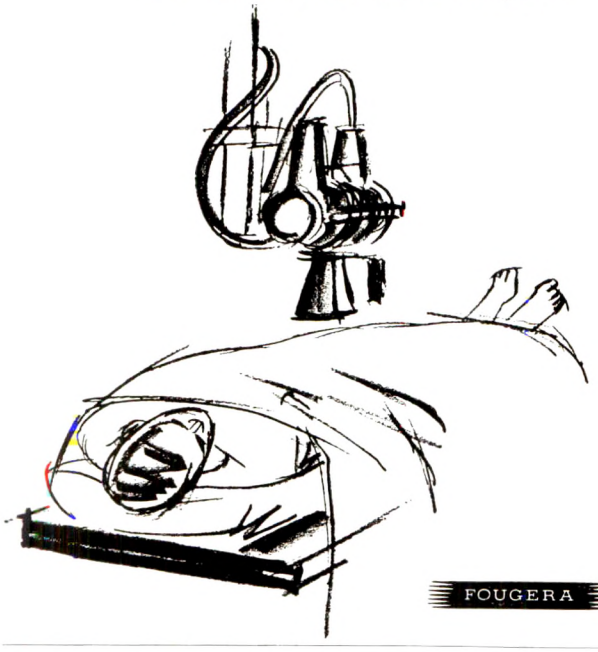
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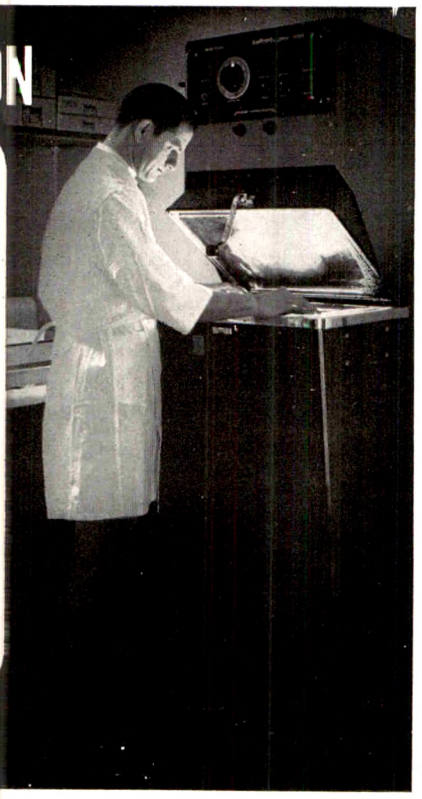
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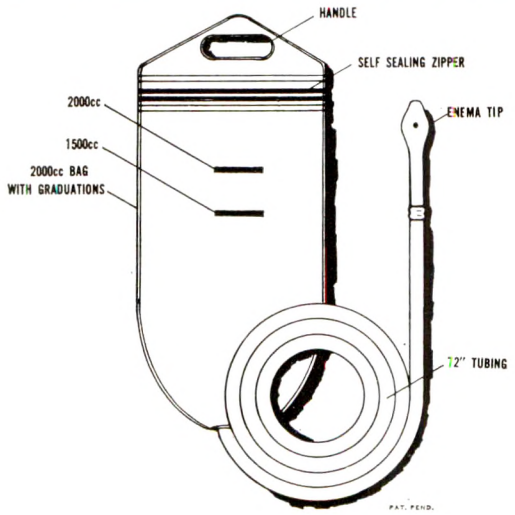
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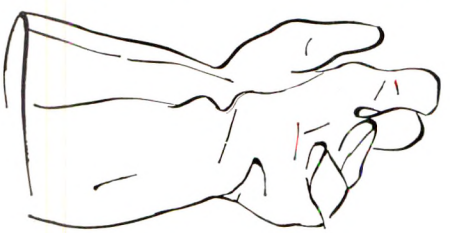
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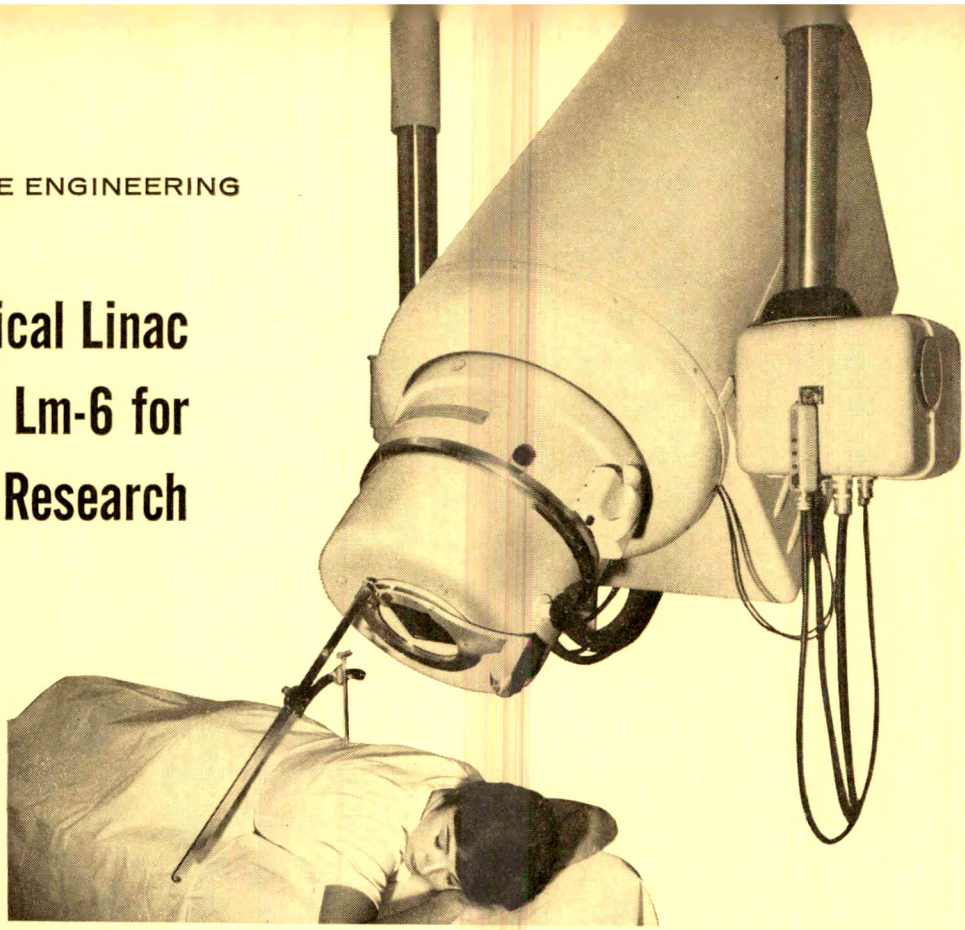
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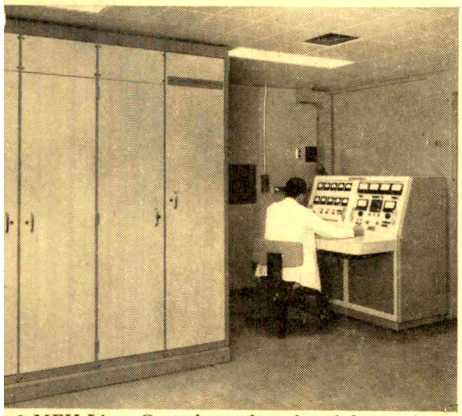
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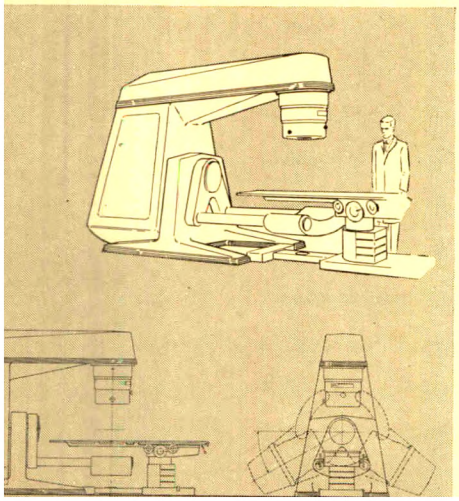


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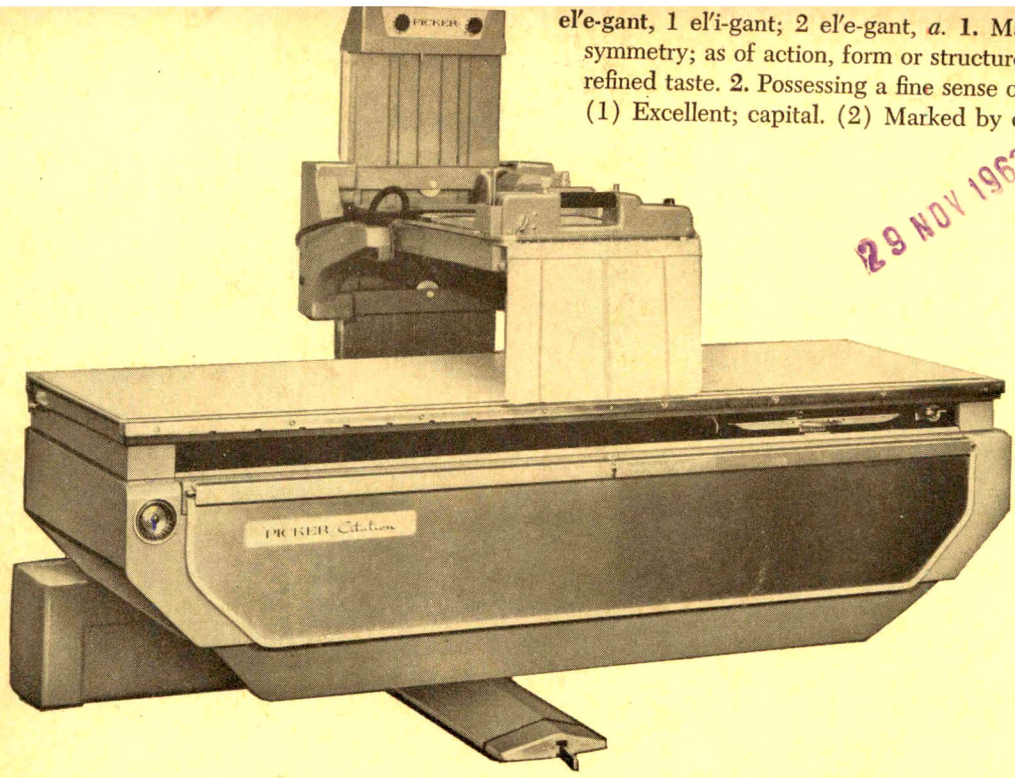
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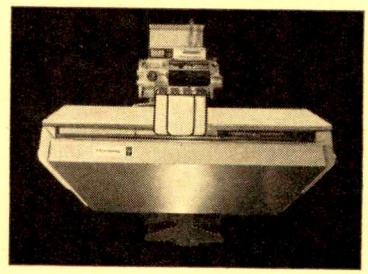
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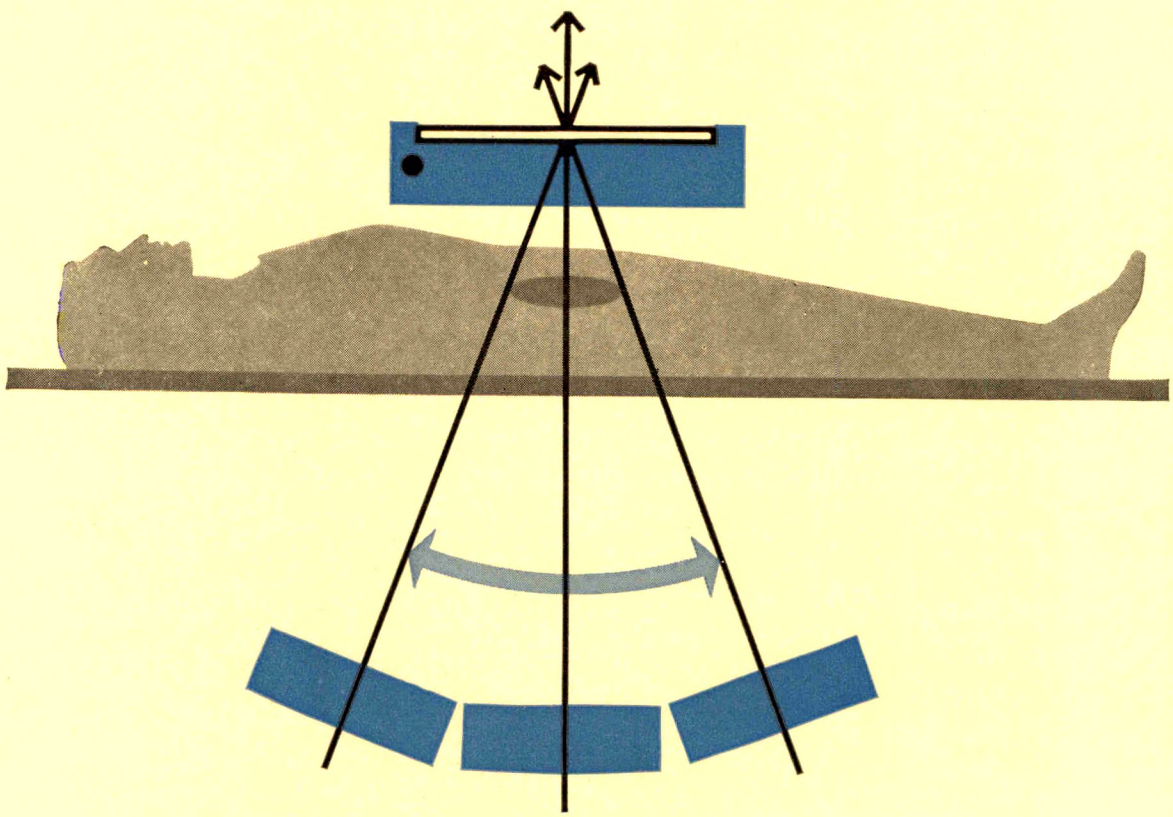
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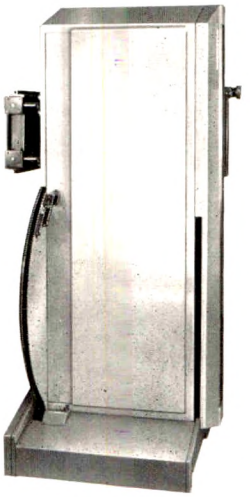
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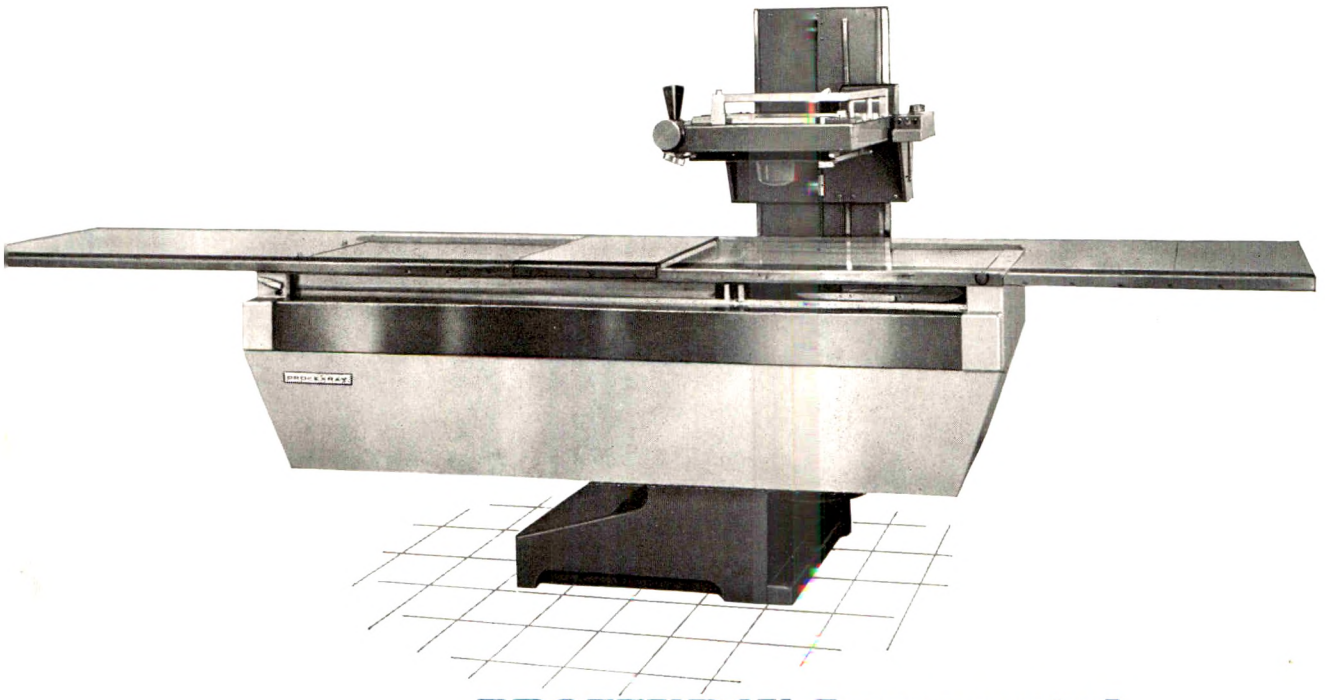


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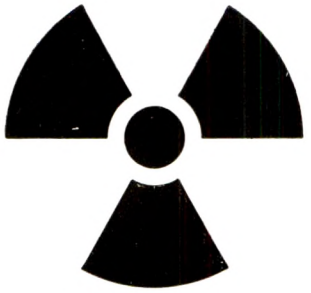
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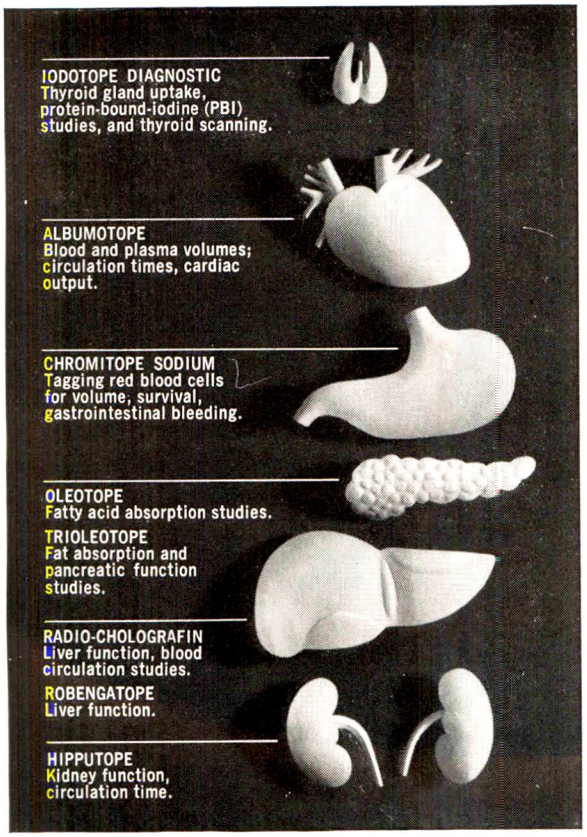
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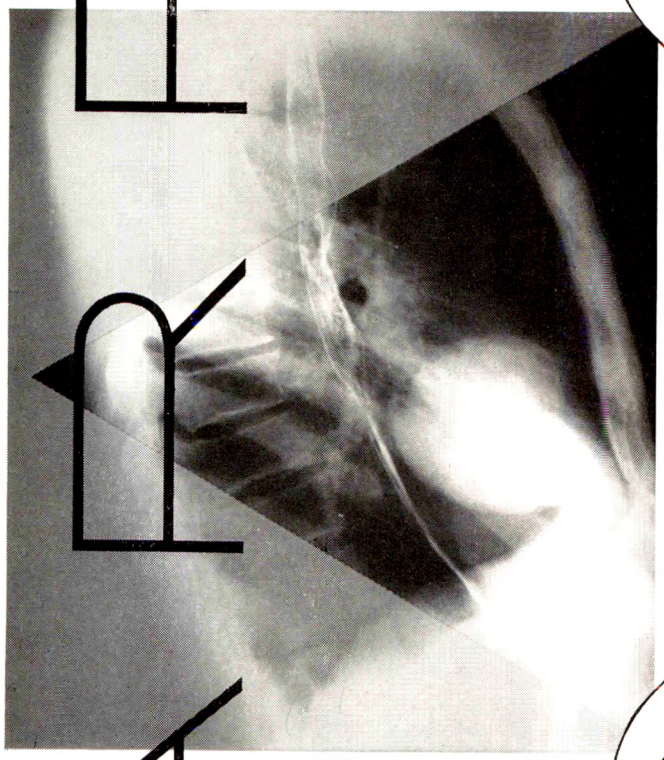
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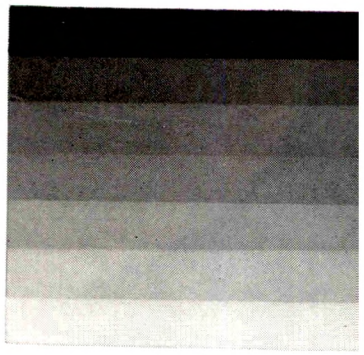
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Ilford X-ray Fixer fixes all Ilford X-ray films quickly and cleanly, with no trace of residual haze. We invite you to test these chemicals against any others available. You will find they materially improve the already fine Ilford X-ray product.

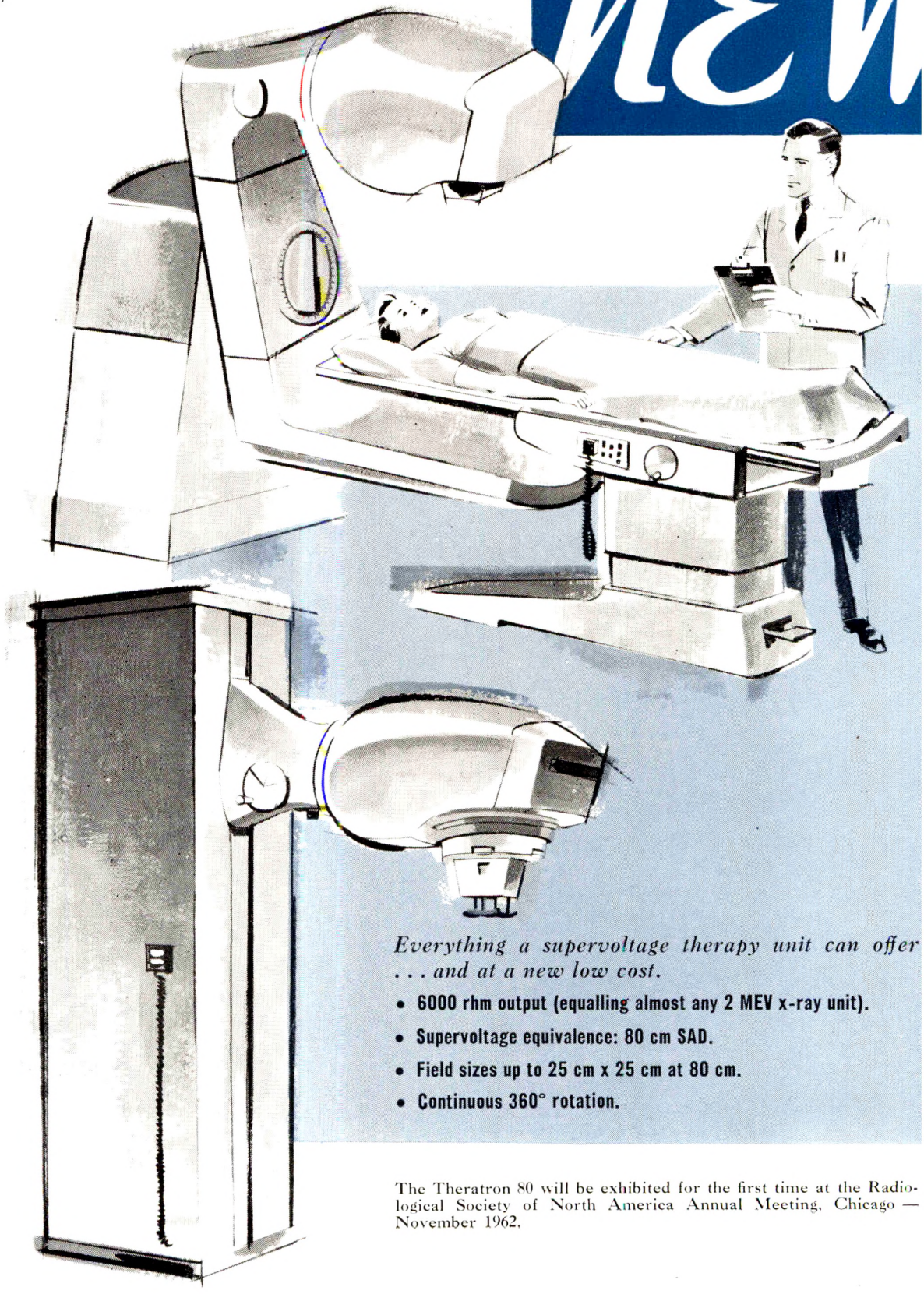
Phenisol Developer, Phenisol Replenisher and Ilford Fixer are supplied in one gallon unbreakable plastic bottles, each yielding five gallons of working strength solution. Order from your Ilford supplier.

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- **6000 rhm output (equalling almost any 2 MEV x-ray unit).**
- **Supervoltage equivalence: 80 cm SAD.**
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- **Continuous 360° rotation.**

The Theratron 80 will be exhibited for the first time at the Radiological Society of North America Annual Meeting, Chicago — November 1962.

rom AECL

the THERATRON 80

he ELDORADO 8

The two latest additions to AECL's entirely new, advanced line of Cobalt 60 Teletherapy Units announced early in 1962.

The THERATRON 80 incorporates all the deluxe features of the most expensive equipment yet it is priced below many units with a lower capacity and shorter treatment distances. A host of advanced ideas for speedy and accurate treatment are incorporated in the design. These, together with an extensive range of precision localizing and treatment accessories make the THERATRON 80 the most desirable Cobalt 60 unit available.

CHECK AND COMPARE:

- Multivane collimator with telescopic penumbra trimmer.
- Choice of final beam definition at 47 cm or 65 cm from source.
- Built-in motorized treatment stretcher.
- Uniform attenuation stretcher top.
- Motorized head swivel.
- Optical treatment field light localizer and distance indicator.
- Optical backpointer.

OPTIONAL ITEMS

- Built-in x-ray localizer.
- Choice of pendulum counterweight design.
- Built-in transit dose monitoring system.
- Pin and arc localizer.
- Digital timer.
- Comprehensive beam shaping system.
- Set of wedge filters.
- Stretcher top with removable sections.

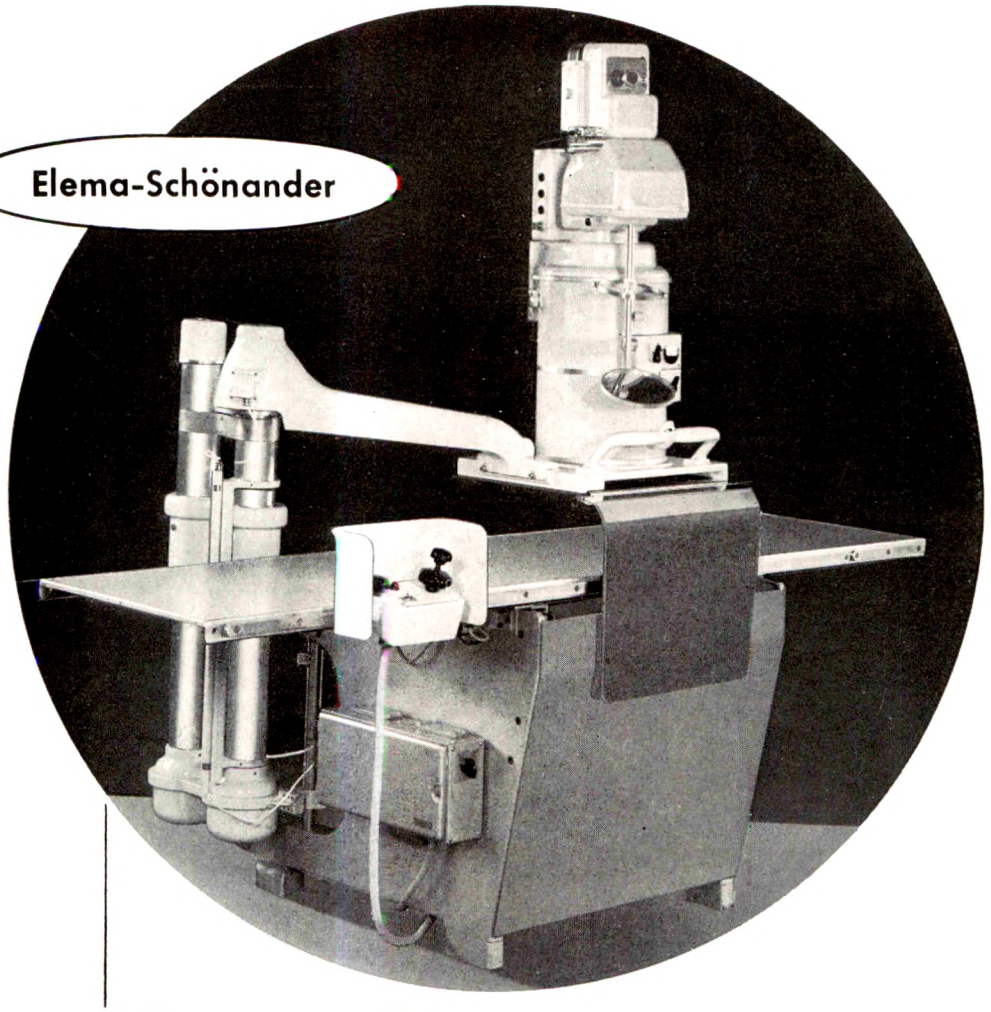
For the full story on the THERATRON 80 and its sister unit for stationary field teletherapy, the ELDORADO 8, please write to the address below. Our representative in your locality will be pleased to call.

Sales and service representation in over 100 countries.

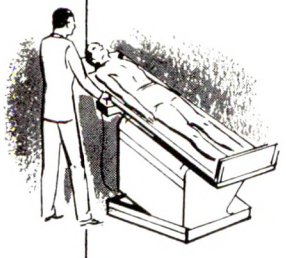
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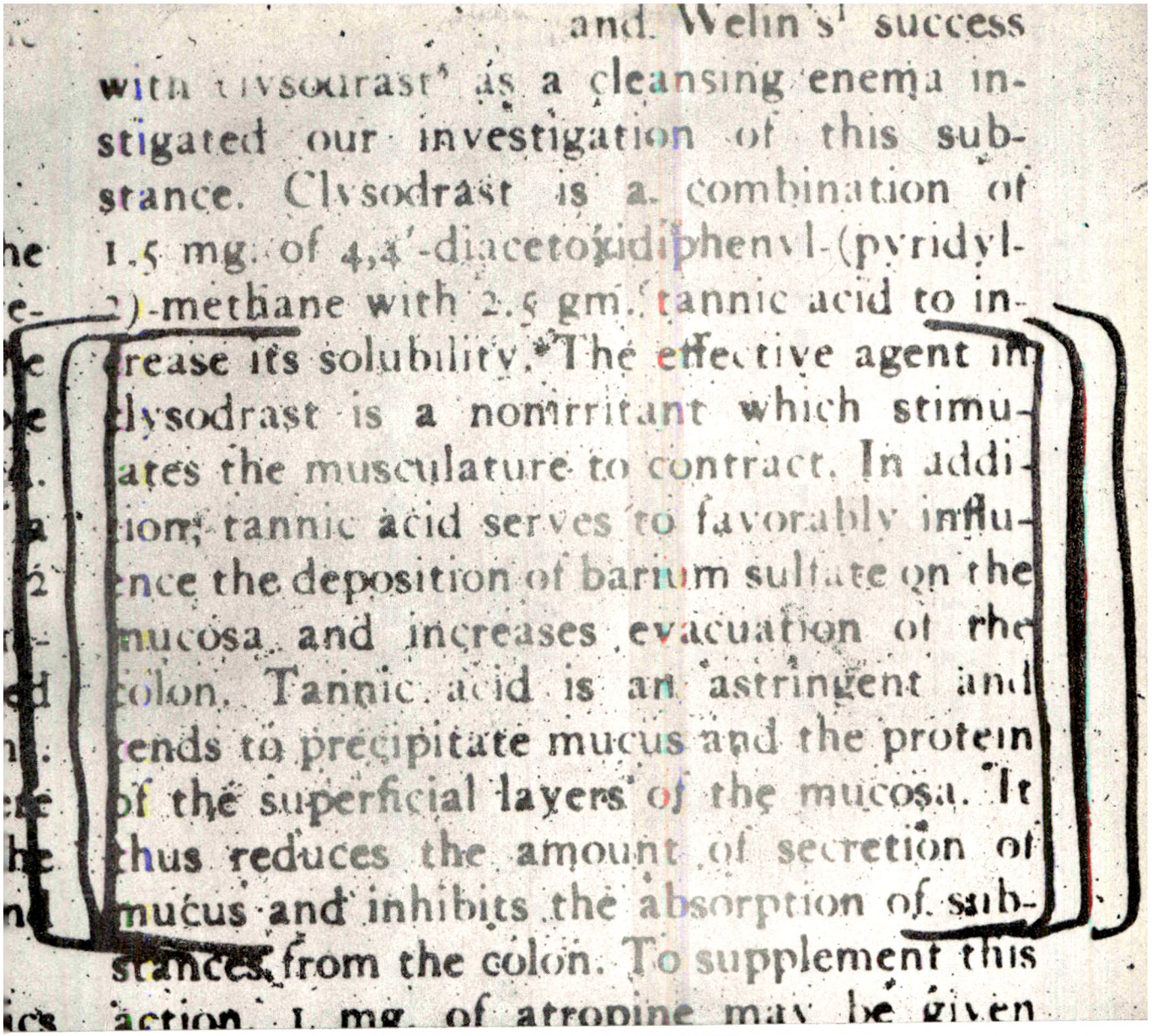


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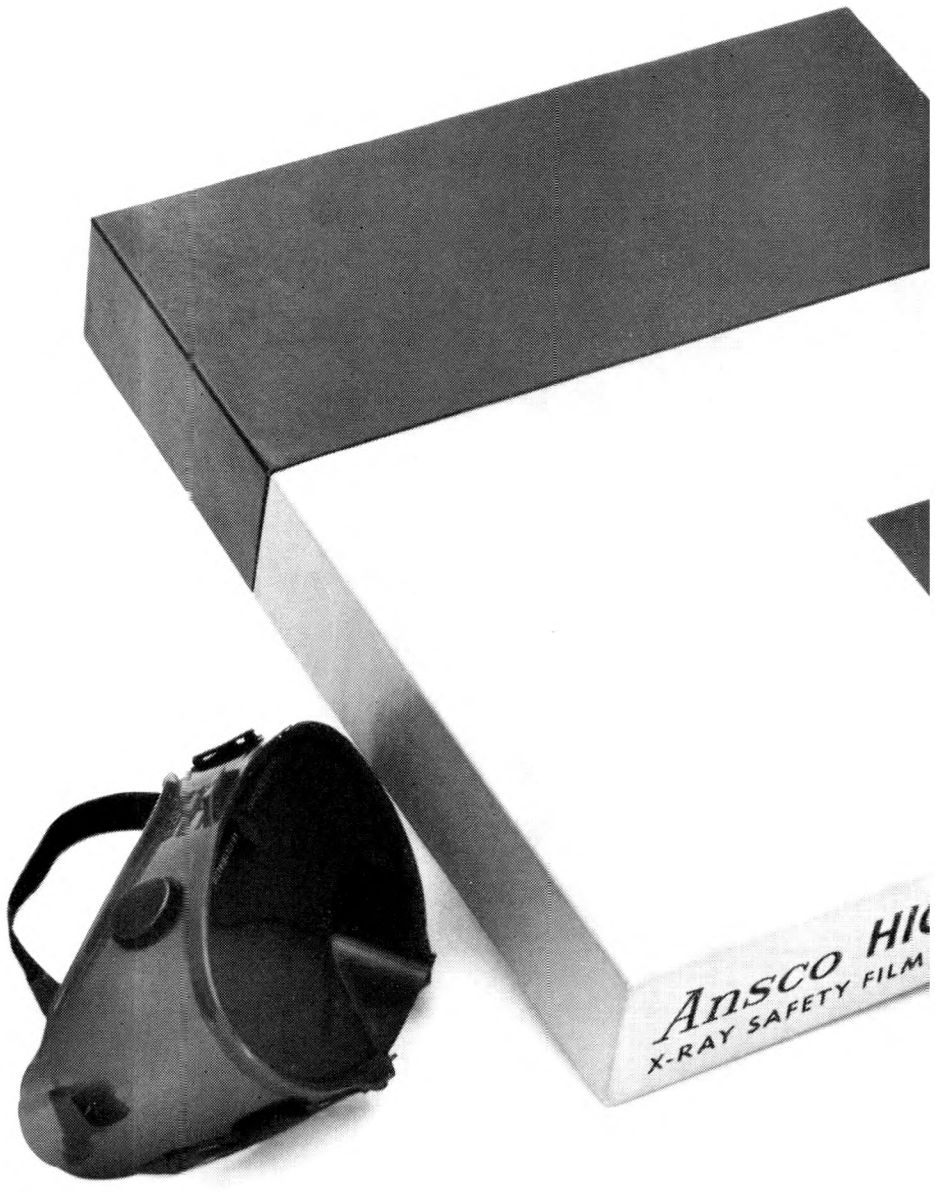


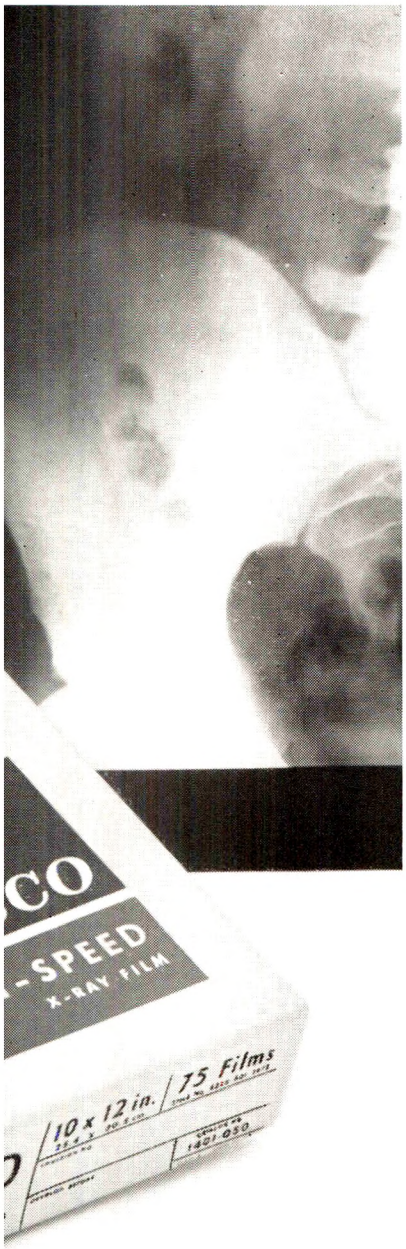
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Literature, sample, and details regarding use of Clysodrast will be promptly furnished by
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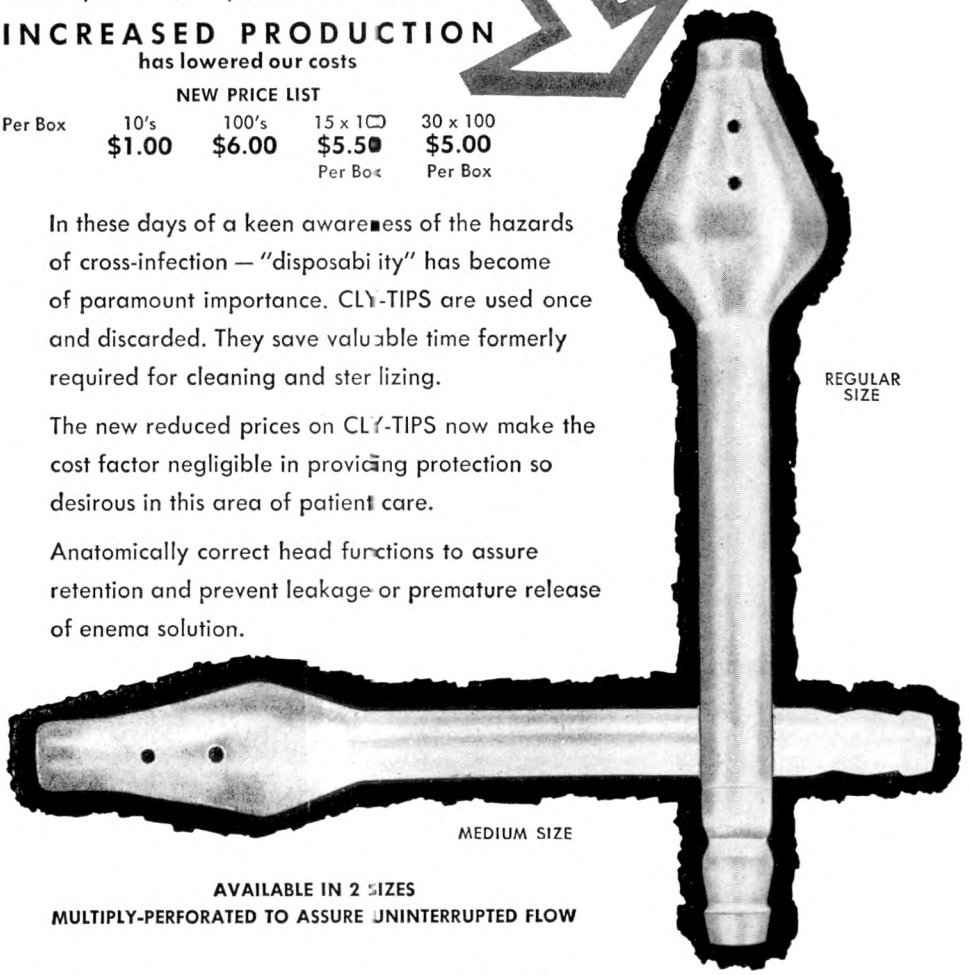
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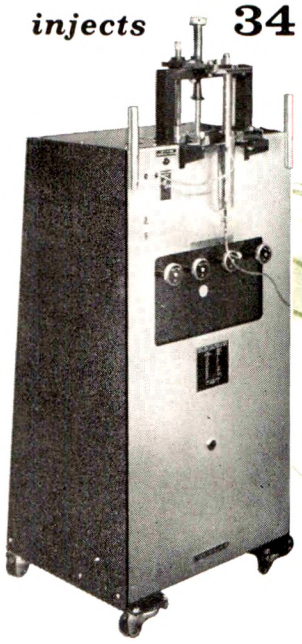
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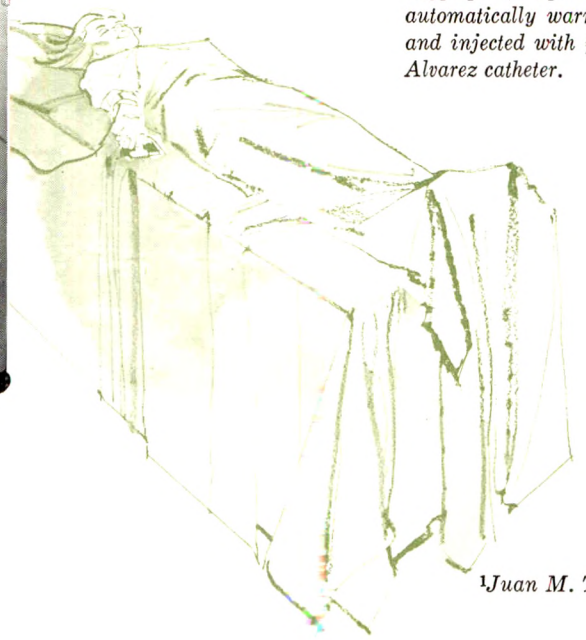
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[1]*Juan M. Taveras, M.D.*

Angiography today calls increasingly for the higher and higher injection pressures available only with a power injector. In cineradiography of cardiac catheterization, for instance, a really high pressure injector can rapidly discharge, with one burst, an undiluted volume of radiopaque into the bloodstream, thus radically reducing the quantity necessary for adequate visualization. When each heartbeat propels the total injected volume as an unattenuated bolus, as little as 10 cc may well suffice.

Because the newly improved Taveras Injector can easily be set to deliver pressures from 75 up to 1075 pounds, it will fully satisfy any foreseeable angiographic demand. Beyond that, it incorporates an impressive array of technical advances[3] dictated by the experience gleaned from literally thousands of angiographic studies.

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[3] **Get the full story** of its technical versatility from your local Picker representative or write Picker X-Ray Corporation, 25 South Broadway, White Plains, N.Y.

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If you feel that your institution can make use of the Gordon Full Size X-Ray Film Projector, we will be glad to have one of our own engineers visit you to discuss and evaluate your situation. We do not manufacture stock models. All of our equipment is custom made for your needs. The cost of these units is from $1,800 to $5,000 with all attachments. We evaluate, sell, install, and service directly . . . not through dealers.

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X-RAY NEWS

No. 58

Oblique view of the lower end of the fibula

Mrs. Roswitha Mohr
Parksville, British Columbia





I would like to describe a view of the ankle joint which, used in conjunction with the routine a.-p. and lateral views, is most helpful in determining any incomplete oblique fracture of the lower end of the fibula. We made this additional view a routine one in our x-ray department and as a result, diagnosed several fractures which could not be seen in either the a.-p. or lateral view.

The purpose of this view is to separate the lower end of the fibula from the tibia and the os calcis. To get this view, the patient is placed in a comfortable position on the table, lying on the side of the injured ankle. The cassette is elevated with sandbags to an angle of 30 to 35 degrees, and the foot is placed on it. (You will probably have to move the foot and the cassette right up to the edge of the table.).

It is particularly important in positioning this view to get the foot at right angles to the lower leg. Otherwise, your radiograph will show the lower end of the fibula overlapping the calcaneus. To avoid this, push the toes upward as far as you can without causing the patient undue pain.

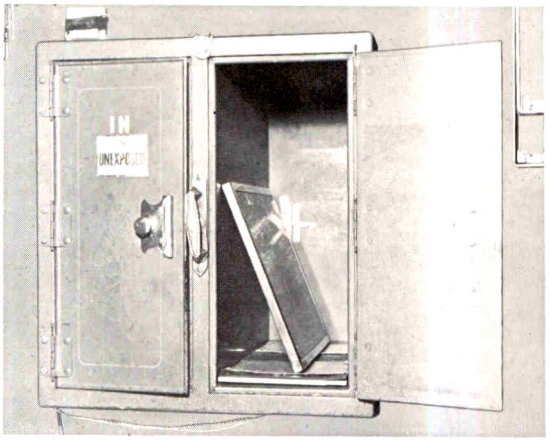
It is equally important to position the lower leg with its lateral aspect almost on the border of the cassette, rather than in the center. This compensates for the tilting of the x-ray tube and thus centers your picture properly.

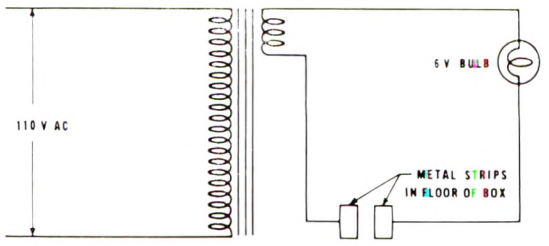
The x-ray tube should be at an angle of at least 30 degrees, to separate the lower end of the fibula from the os calcis. At 30 degrees off vertical, the central ray will enter the ankle at the level of the internal malleolus, but between the internal malleolus and the back of the leg. This should give you adequate separation. The tube-cassette distance used in the radiograph shown here was 27 inches. You can see in the photograph the relationship between the tube, patient and cassette.

This ankle view is particularly valuable in x-ray departments of small rural hospitals, where no radiologist is available and the physician depends on his own interpretation (or that of the x-ray technician!). And it helps where fast action is needed, such as, hospital admission and treatment or surgery. In my own experience many doctors (nonradiologists) particularly appreciate this view because in negative cases they are never left with that unpleasant feeling of possibly having missed a fracture. In positive cases, of course, this additional view helped them to determine the extent of bone damage and the degree of separation of the fragments. And in most of these positive cases it was not advisable to delay therapy several days until a qualified radiologist could report by mail.

OVER

Tips for Technicians





Pass box lamp helps keep film moving

by **Lawrence A. Davis, M.D., Radiologist**
and **Robert Adams, Chief of Maintenance Department**
Children's Hospital, Louisville, Kentucky

"How can we keep exposed film moving quickly into the darkroom?" This was a nagging problem in our busy x-ray lab. Film tended to pile up in the pass box. Technicians just couldn't take time out to check the box as much as they should.

Our solution was to rig a simple circuit connected with a red lamp that indicates when new cassettes have been deposited in the pass box.

Here's how it works. The small six-volt lamp is in series with one of the lines attached to two metal strips that have been mounted on a piece of thin plywood on the floor of the "exposed" section of the pass box (see diagram above). These strips have been placed several inches apart.

When the steel-rimmed cassette is placed in the box, it touches both metal strips, completes the circuit, and lights the lamp in the darkroom.

The lamp has been painted with dark red fingernail polish to cut down the light intensity. Power comes from a simple doorbell transformer mounted above the box and tied in with the regular house current.

Inexpensive tank reinforcement for silver recovery unit

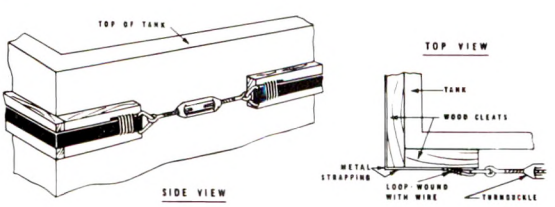
by **Arthur C. Barnes, Wrentham, Mass.**

In the use of a silver recovery unit in the X-ray department I have found it desirable to keep this equipment in a molded 15 gallon tank out on the floor of the darkroom.

Lack of support for the tank walls has in the course of time made considerable bulges because of the weight of the tank contents. Eventually this would cause the walls to crack and leak discarded fixer. But this condition can be prevented easily and inexpensively with some scrap metal straps and hardware that cost less than a dollar.

Wooden cleats a half-inch wide and two inches thick are placed about ¼, ½ and ¾ the distance from the bottom of the tank. Old metal strapping, of the type used to reinforce large corrugated cartons, is placed around the tank and over wooden cleats and then fastened with simple turnbuckles of the size normally used on screen doors. The turnbuckles are tightened to bring the sides of the tank back into shape and prevent further bulges.

The sketch below shows how this is done. It permits you to use a far cheaper tank than usual and to extend the life of such a tank—as well as preventing any sudden, disastrous leaks that could flood your floor with used hypo.



E. I. du Pont de Nemours & Co. (Inc.)
Photo Products Dept., Wilmington 98, Del.

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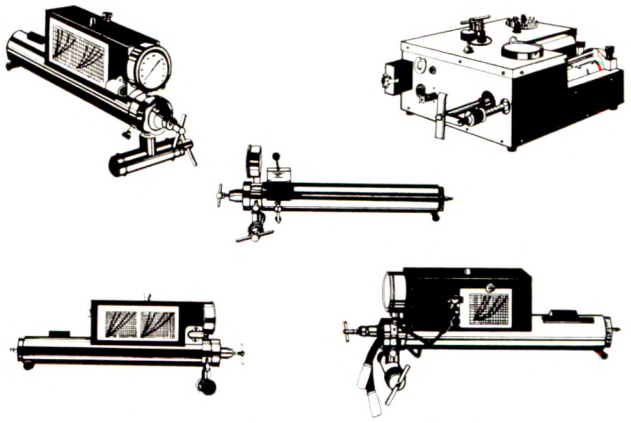
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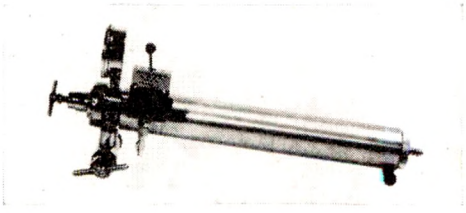




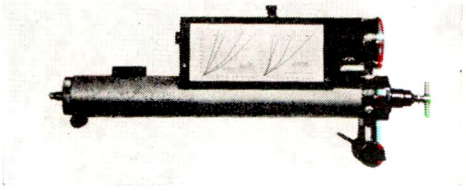
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ORABILEX provides a more certain diagnosis with less toxicity and fewer side effects.

1. Lasser, E. C., et al: J. Nuclear Med. 2: 79 (April) 1960. 2. Sachs, M.: Personal communication. 3. Whitehouse, W. M., and Fink, J. E., Jr.: Univ. Michigan Med. Bull. 25: 238 (July) 1959. 4. Morgan, L. A., and Parks, R. E.: Radiology 74: 436-441, 1960. 5. O'Laco, J. P., and Wilcox, W. A.: Wisconsin Med. J., 59: 249 (April) 1960. 6. Reeves, R. J.: Am. J. Roentgenol., Rad. Ther. and Nuclear Med., 83: 843 (May) 1960. 7. Farrell, T. E., and Weldon, W. V.: Southern Med. J., 54: 54 (Jan.) 1961. 8. Tatelman, M., and Steel, W. B.: J. Michigan State Med. Soc., 60: 357 (March) 1961.

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Fewer repeats... because the reliability of ORABILEX as a diagnostic medium is such that non-visualization usually indicates the presence of hepatic or biliary disease (unless disorders of the small intestine interfere with absorption). Fewer repeats mean greater economy.

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No need for double-triple dosage... because a single 4.5 Gm. (6 capsule) dose of ORABILEX provides more opacifying material for distinctly better visualization. Should non-visualization occur and the physician wish to secure his findings, one additional 4.5 Gm. dose may be given for re-examination the following day.

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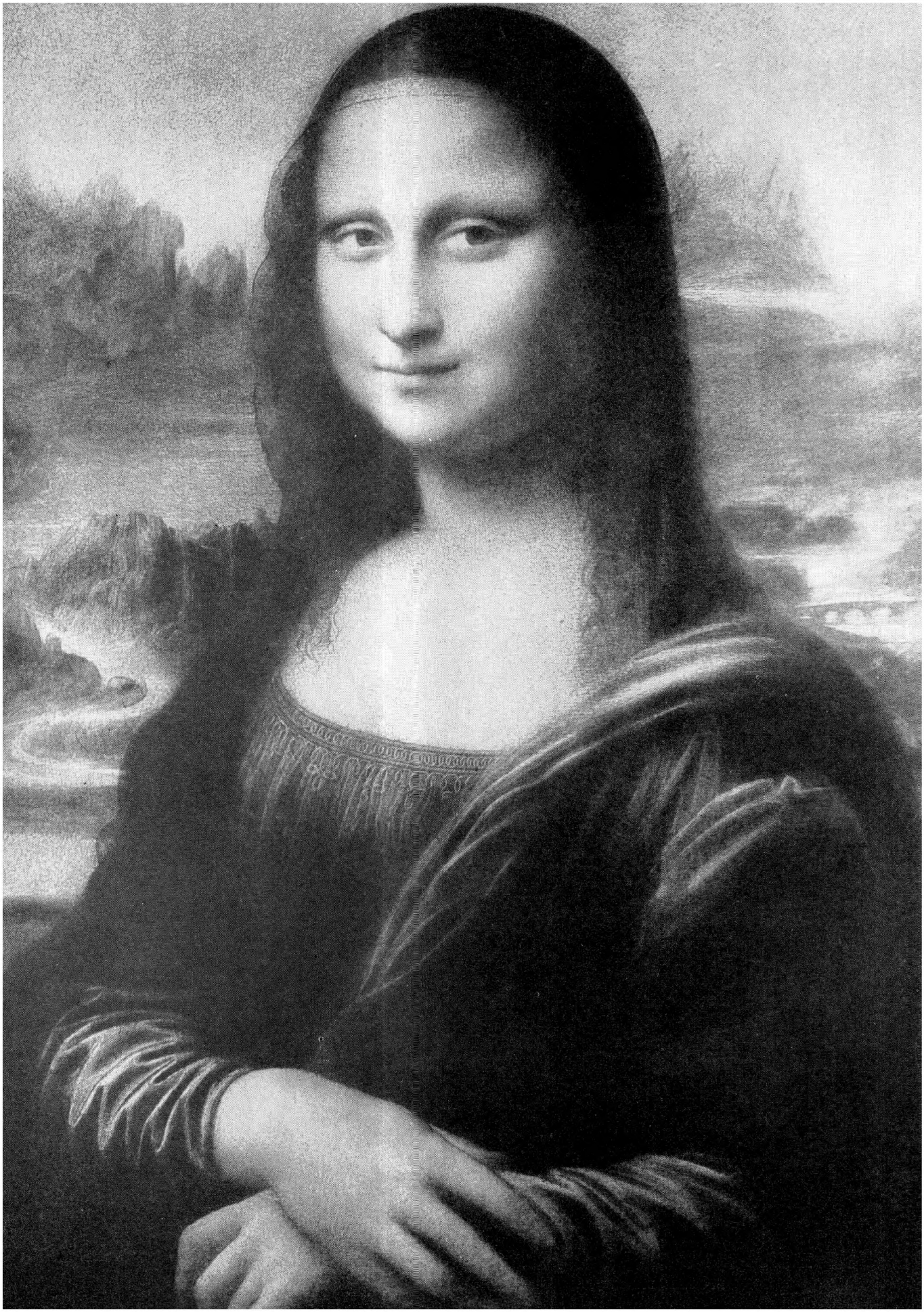
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Cerebral Angiography: "Cerebral angiography by the carotid route is now relatively safe, thanks to newer contrast media like *Hypaque,* the risk of complications being less than 3 per cent."[3]

Operative Cholangiography: "Hypaque sodium has been very satisfactory."[4]

Hysterosalpingography: Hypaque sodium 50 per cent solution "... provides satisfactory visibility both at fluoroscopy and in films ... currently considered most satisfactory..."[5]

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References: 1. Lloyd, F. A., and Cottrell, T. L. (Northwestern Univ.): Diatrizoate sodium, *Ann. New York Acad. Sc.* **78**:987, July 2, 1959. 2. Lance, E. M., and Killen, D. A. (Vanderbilt Univ.): Experimental appraisal of the agents employed as angiocardiographic and aortographic contrast media. I. Neurotoxicity, *Surgery* **46**:1107, Dec., 1959. 3. Pool, J. L. (Columbia Univ.): Early treatment of ruptured intracranial aneurysms of the circle of Willis with special clip technique, *Bull. New York Acad. Med.* **35**:357, June, 1959. 4. Davis, D. R. (St. Luke's Hosp., Kansas City, Mo.): Operative cholangiography by utilizing the cystic duct, *Surg. Gynec. & Obst.* **112**:378, March, 1961. 5. Youngstrom, Karl A. (Univ. of Kansas): Study of use of diatrizoate sodium (Hypaque), *J. Kansas M. Soc.* **59**:308, July, 1958.

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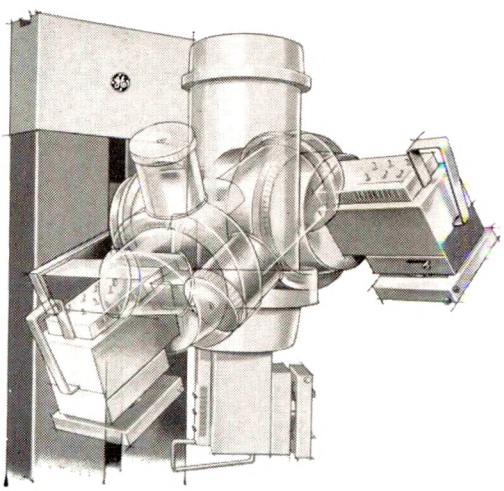
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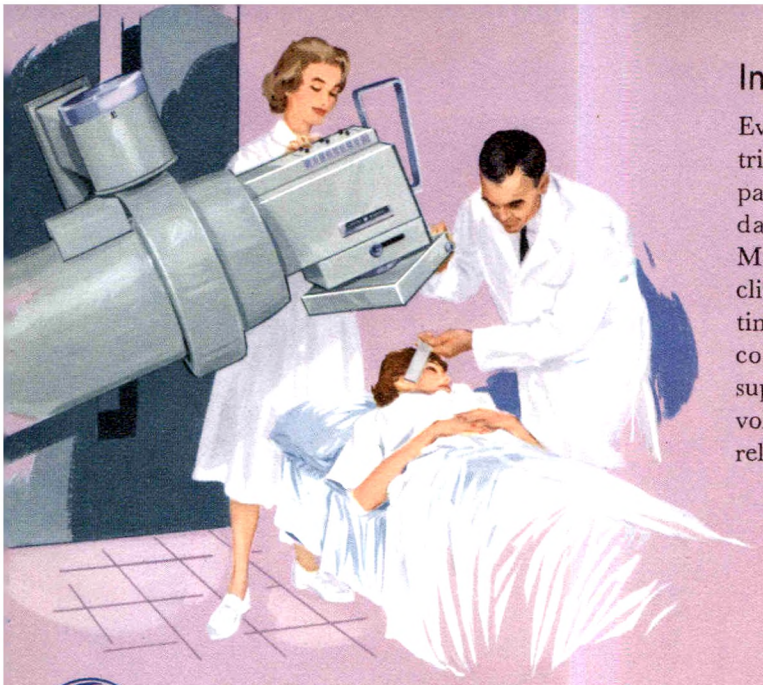
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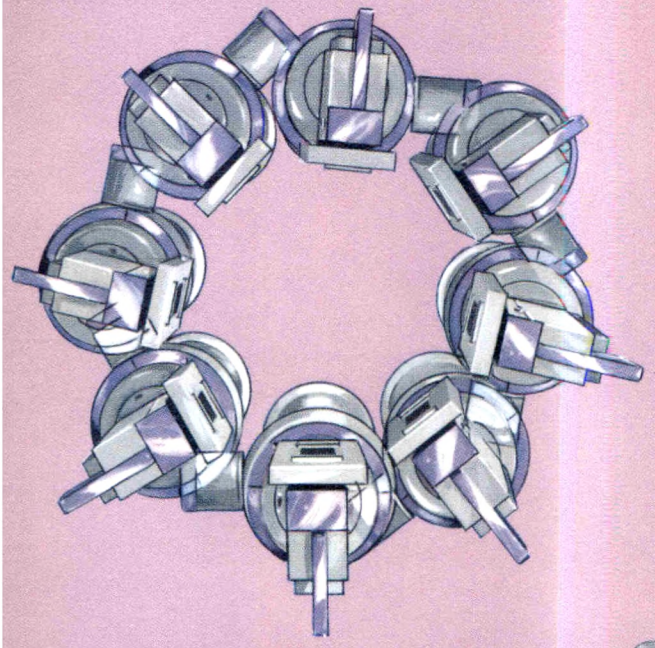


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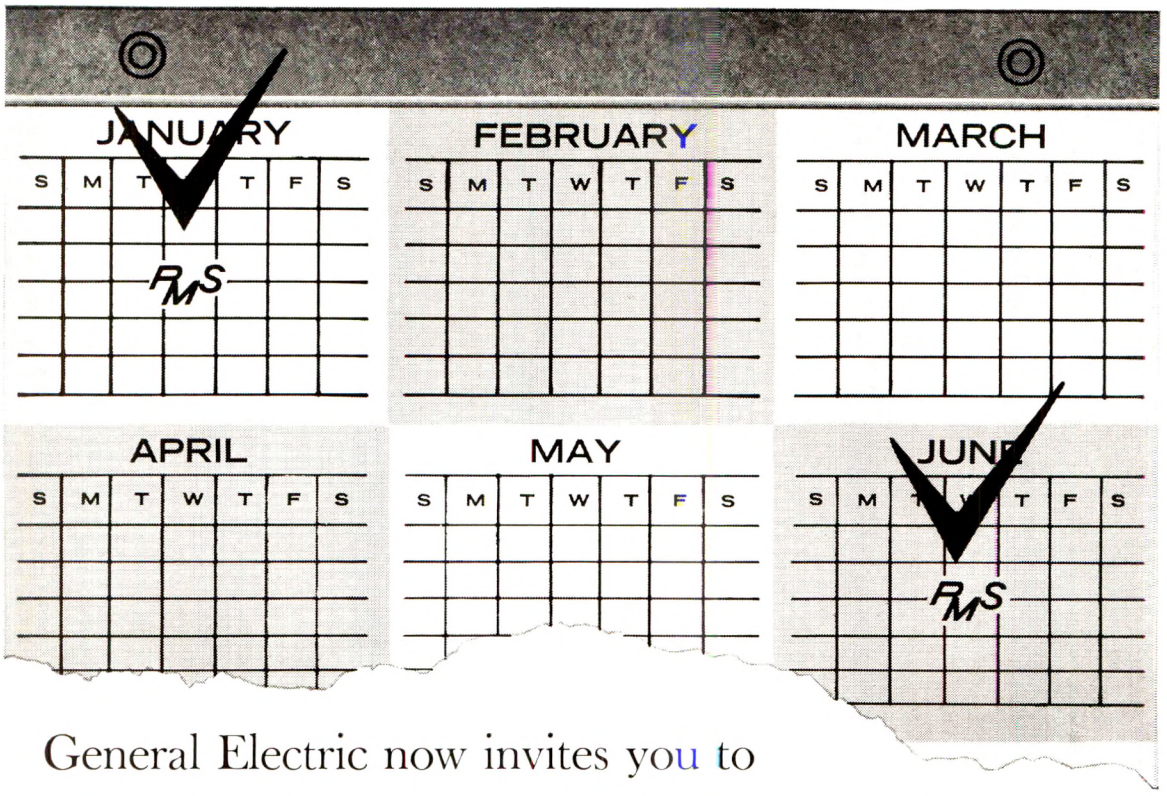
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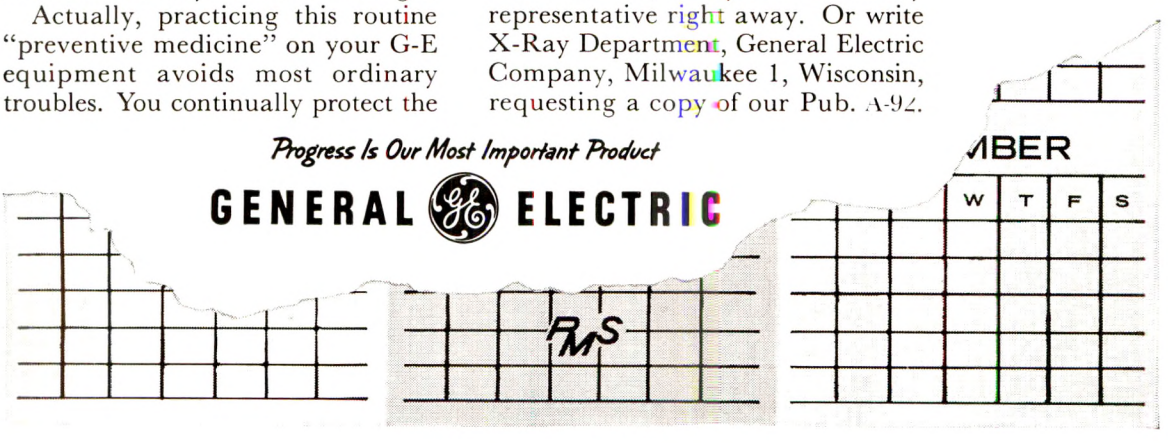
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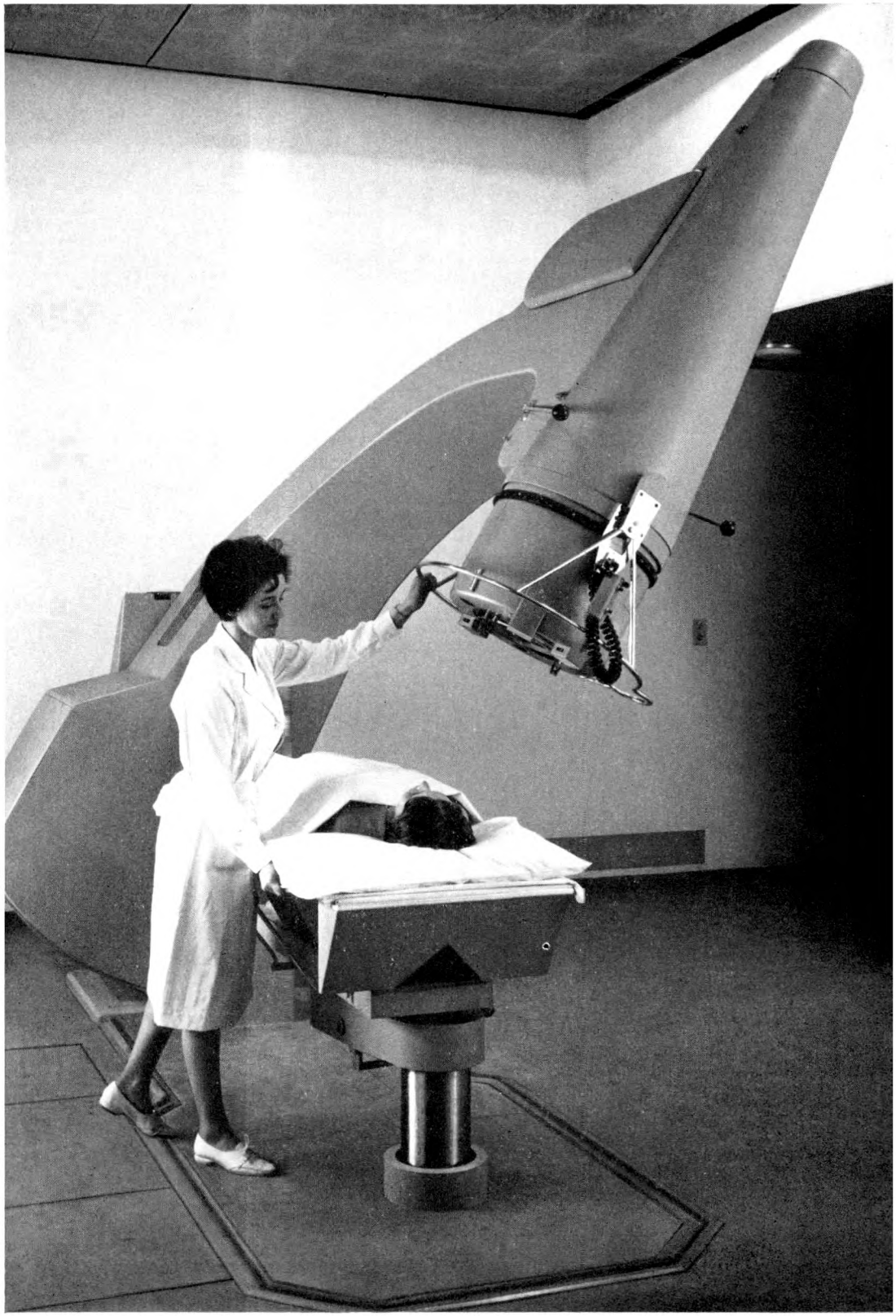
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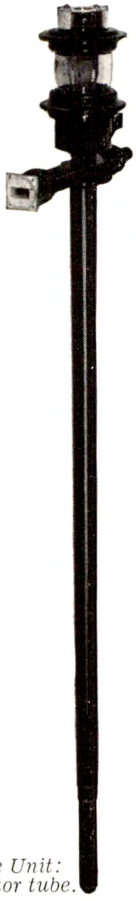
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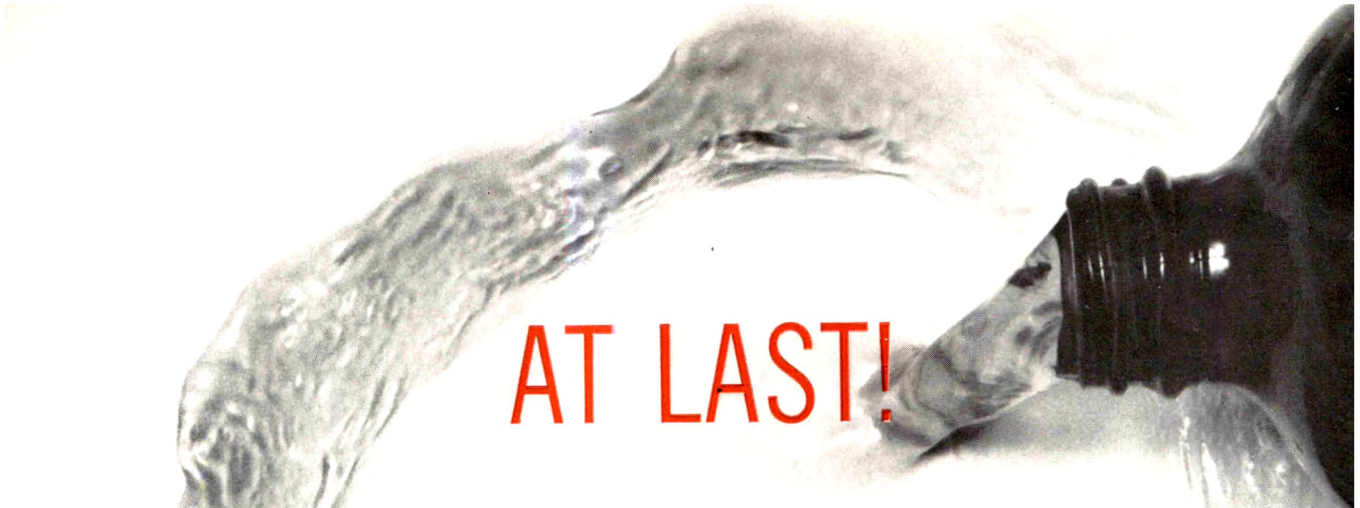
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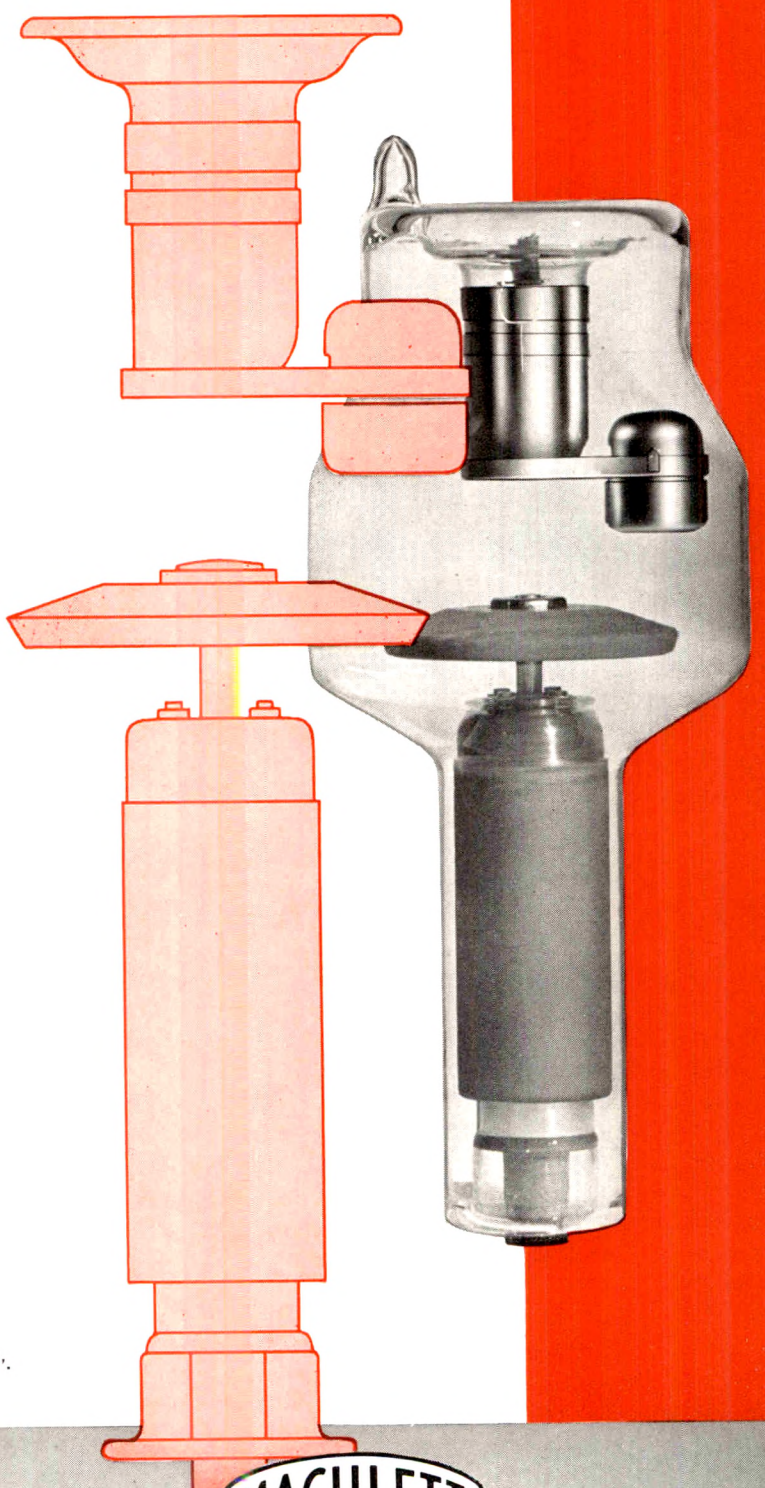
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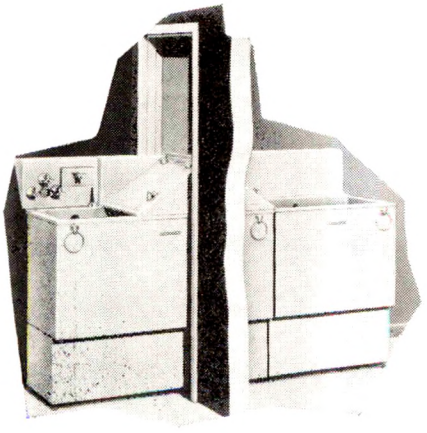


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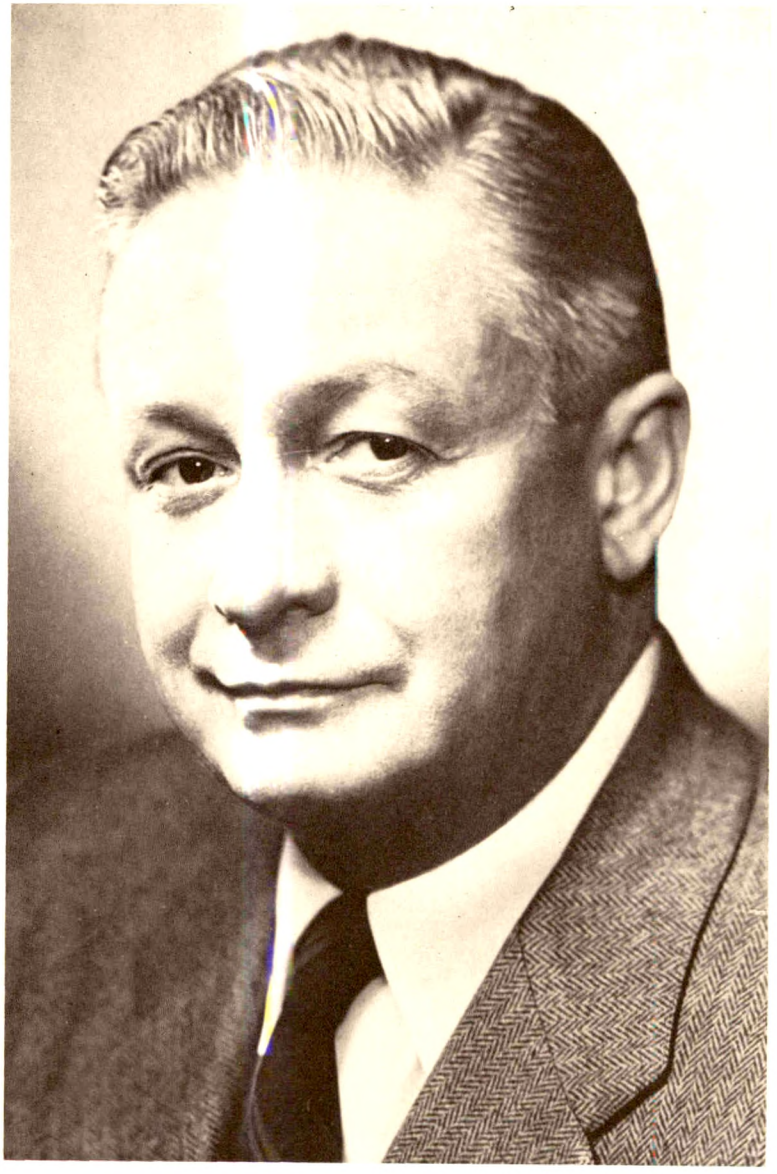




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LAURENCE L. ROBBINS

Hickey Lecturer, 1962

UNIVERSITY

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 88 SEPTEMBER, 1962 NO. 3

PROBLEMS IN THE EDUCATION OF RESIDENTS IN RADIOLOGY*

HICKEY LECTURE, 1962

By LAURENCE L. ROBBINS, M.D.†
BOSTON, MASSACHUSETTS

Preston Hickey, one of the foremost educators in Radiology, did more to promote the future of Radiology in this section of the country than most of us realize. I have known a large number of residents trained by him, and have been profoundly grateful to have had this perception of Dr. Hickey. When Dr. Eyler extended the invitation to me to give this lecture, I was overwhelmed and have remained so! One has only to review the list of previous Hickey lecturers to appreciate that this honor can only be accepted with pride and humility.

May it be said here that Hickey was a remarkable educator. Therefore, I have chosen to concentrate on some of the major problems in the education of residents in radiology—a subject in which he was extraordinarily versed—with the intent of amplifying some of his own ideas which were so worthy that they are still in effect today. In reviewing the life of Preston Hickey, it was possible to identify oneself with his problems as the son of a minister; I could also recognize some of his problems as an educator. Beyond these similarities, there is dissimilarity, for he has done far more than I can ever anticipate doing for Radiology. In his work he demonstrated the virtues of a great educator as: (1) An outstanding Radiologist at Harper Hospital; (2) Founder and Editor of one of the great journals of the specialty; and (3) Teacher, professor, and an honored member of the Faculty of the Detroit College of Medicine and later of the Faculty of Medicine of the University of Michigan. It is a unique opportunity to be able to dwell upon his ideas and ideals which I believe are now being extended to the present and future.

DR. EYLER requested at one time that I discuss the future of Radiology. I deferred in using the exact terminology and hope to apply the contention that our greatest potential in the future of Radiology is the Program in Resident Education. Since the medical schools seem to expose the student less and less to the scope and potentialities of Radiology, it becomes

* Presented at the meeting of the Wayne County Medical Society, the Wayne State University College of Medicine and the Detroit Roentgen Ray and Radium Society, Detroit, Michigan, March 1, 1962.

† Clinical Professor of Radiology, Harvard Medical School and Radiologist-in-Chief, Massachusetts General Hospital.

necessary that major education of the radiologist start in the internship and in detail in the Resident Training Programs. In the combination of the educational program and the resident lies the future of Radiology.

It is proposed in this discussion of education of residents to review (only as personal opinions) the position of the American Board of Radiology, to review the importance of the Residency Review Committee, and hopefully to stimulate thoughts to current or future problems of the specialty which must be met and faced as they occur. You may ask why we should discuss the education of the resident in Radiology at a time when education is at one of its highest and most progressive levels. My only answer would be—it is not enough. When the term "education" is used, it is not directed totally toward teaching, but it also includes the oft unrecognized necessity of learning. Many try to teach, few are outstanding, and the student remains at the vertex of the problem. If opportunity for learning is not available, then the teacher is a failure. Reluctant to say this, I believe that a number of radiologists are overly superior, self sufficient, and possibly afraid of the future—the younger man is considered inferior and incapable. Many of my friends believe that a young man just out of his residency is not to be trusted in a responsible position and surmise that he knows little of the basic responsibilities of the specialty. I contend, however, that these young men are the most knowledgeable in the field. Actually, if you really want to know the answer to a medical problem, what better source of information is there than the alert fourth year medical student? They are the ones who have absorbed recent important medical knowledge and they have the ability to think and to reason. Some of us older people may have better judgement regarding a particular problem, but the best care of the patient may well come from the young man or woman just out of training. Those in the specialty of Radiology can be elated and obligated for the residents in Radiology.

It is still the necessity of the educators of future radiologists to recognize possibilities for improvement. Therefore, one must investigate some of the various forces involved.

INTRINSIC FACTORS

BASIC EDUCATION

The basic source of residents is the medical school. At graduation 84 per cent of the class have decided on their future activities, and during internship only about 11 per cent change their minds. Therefore, if we are to interest bright young men in Radiology, it must be a part of the medical school curriculum. Radiology should be presented to the medical student largely as an attraction and practically only as a required subject. The former is most important, but the student should be exposed to basic possibilities, the limitations and the effects. This entails scholarship and research which Radiology has. It can also be stressed as one of the most important factors in the care of patients. The seminar type of presentation in the integrated teaching relationships should prove to be one of the most fruitful for induction of the medical student into the values of Radiology.

THE RESIDENT

One can become involved in a long discussion as to whether the American Board of Radiology is an educational institution and should determine policies or whether it should be excluded from the field of education. I shall not try to defend the status of the Board but to point out the fact that, as far as postgraduate education in the field of Radiology is concerned, the American Board of Radiology is one of the most influential forces.

The Residency Review Committee is nearly as potent in its function of implementing the principles of the American Board of Radiology.

The curriculum and the teaching effort

must originate from the Faculty. It is disappointing that very few teachers have contributed any useful variations on the pattern of teaching since the day of Hickey which might lead to progress in the field of Radiology.

EXTRINSIC FORCES

Here we must recognize certain present day developments that are forcing themselves upon us to a greater and greater degree. These are in certain instances the effect of Blue Cross and Blue Shield, Labor Organizations, or other third party payment arrangements and also the increasing influence of the government on the training of persons in medicine.

A. We are all cognizant of the serious intervention of the insurance companies on training programs. Very few if any of these companies seem to recognize that residents advancing in a specialty are far better trained than the person having only one year of internship. It is unfortunate that the opening of an office after one year of internship indicates to the insurance company that a person is in the practice of medicine and can collect fees from patient or company without argument. How much longer the insurance plans can continue in this direction is a moot question. The American Medical Association and most teachers realize the inequities but so far seem to have accomplished little.

B. Even without socialized medicine, there is bound to be a greater extension of government control into training programs. Government agencies decide what they will support! The graduate in Medicine (in distinction to the Ph.D.) with a long unsubsidized educational period is influenced by the need of financial help usually unrequited by hospitals. Whether these programs are educational with the intent of benefit to the public, or are merely intrusions on present educational principles, is a questionable point.

The position of the private university regarding academic freedom has been well stated by President Pusey of Harvard and intervention of this basic principle cannot be tolerated. The position of the educators in Radiology can be no less firm.

The various Government agencies are not interested, according to their statements, in training clinicians in Radiology or most other branches of Medicine. They are interested in training academic and research individuals as directed by Congress, but the emphasis on research has become extremely exaggerated. If there is a need for government influence in the field of Medicine then it is in construction of additional medical schools and in assisting medical students and providing proper subsidization in later training without direct stipulations on curriculum. There is a great question in most educators' minds whether all of the emphasis should be placed on research. There is full agreement that there is need for research in all branches of Medicine, but it is also our obligation to see that good clinicians as well as academicians, researchers or other investigators are trained properly.

THE GENERAL EDUCATION PLAN

Now as to some of the actual problems of the resident (excluding marriage, families, finances and military service)—who is to determine the policies concerning his education? At this stage, rightly or wrongly, they derive from the American Board of Radiology.

AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology recognizes programs which provide training in the entire field of Radiology and, as of July 1, 1962, this includes nuclear medicine as a mandatory portion of the examination. A program now approved for 3 years' training in Radiology can provide a curriculum in diagnostic radiology or in therapeutic radiology. In the future it may well be that divided programs in diagnostic radiology and radiation therapy each requiring 3 years of training may be approved. There will be further discussion of these possibilities later. In addition to the formal training period, the American Board of Radiology requires a one year interim

period before the candidate can take the examination.

Why? This period provides opportunity to observe the resident's professional ethics as well as to learn how he applies his newly acquired knowledge. Also it frees him from intensive study during the final months of formal training when he should be broadening his experience. Finally, it is in keeping with most other specialty boards.

How should the young radiologist spend this year? He can use this year in any way he wishes as long as it pertains in some way to his future work in Radiology: practice, further training, teaching, research or military experience in Radiology.

This concludes the required formal training of the radiologist. Some say this completes the education of the radiologist. Nevertheless, it is my sincere hope that this is a false statement! Any radiologist worth his salt, although probably better informed about the entire specialty at this moment, is now at the foot of the stairway in his lifetime of continuous education. Price's Law in distinction to Parkinson's points out that scientific factual information doubles every 10 years. The educator or the practitioner in Radiology has only started to learn, and therefore this is the beginning.

RESIDENCY REVIEW COMMITTEE

How are the policies of the American Board of Radiology activated? These are largely carried out through the Residency Review Committee. The Residency Review Committee is composed of the officers of the American Board of Radiology and 4 radiologists appointed by the Council on Medical Education and Hospitals of the American Medical Association, as well as an ex-officio secretary from the Council. This Committee delves into each training program, and it may be said that each program is reviewed in great detail. Some of the things which the Committee considers very carefully are:

1. The Statistics Presented. It should be added that the Committee is rather astute in picking out fictitious figures.

2. The Program Outline. The curriculum which is to be followed by a resident. What are the intents concerning his real education? Is it likely to be service or education?

3. The Faculty. Are there sufficient supervising radiologists to conduct an educational program, or is the young physician to be carrying out some of the needs of the Department in substitution for an experienced radiologist?

4. The Library. An adequate departmental library as well as a much more comprehensive hospital library must be available.

5. Research. Is there research activity in the Department? Does the resident participate?

These are the more important of many detailed factors which the Committee studies thoroughly.

There have been numerous comments that the Residency Review Committee is not strict enough or that it is entirely too stringent in requiring much more than the preceptor believes necessary. It should be noted that both of these opposite opinions could be subjected to well deserved debate. Nevertheless, having served on the Committee for 4 years and terminating in the near future, it is my strong belief that it is working in the right direction, that it is trying to strengthen programs, and that its aims are centered on better education of the resident. All of these activities imply better care for the patient and the public. I have the feeling that the Residency Review Committee is one of the strongest and greatest forces in the education of radiologists. It is to be recognized that it is largely an activating force of policies which are laid down by the American Board of Radiology; however, its actions, functions and decisions are of huge importance to you and to those whom you are training.

Most of that which has been said implies your obligations to the resident, but beyond these there remain other responsibilities. Perhaps as important as any are your com-

ments to the American Board of Radiology concerning an applicant.

(a) It is true that most preceptors (including myself) believe that any resident carried to completion of his training is a well qualified candidate for examination. Your fair evaluation is, however, of greatest importance to the Executive Committee of the American Board of Radiology. Your failure to answer these inquiries may deprive a resident of the opportunity to take the examination.

(b) It is suggested that you sign these recommendations. An unsigned letter means nothing as anyone can type a letter.

(c) Are the applicant's ethics at the highest level? Has he accepted other appointments and deserted them to accept yours? Are you ready to acknowledge?

The American Board of Radiology has to depend on you to be as fair as possible in your evaluation of a candidate. It must remain assumed that you will not endorse a resident who is unable or who does not desire to give a good account of himself.

COMMUNICATIONS

Communications are of importance not only to the resident but to the preceptor, and I shall be the first to say that they should be improved. The present methods by which the American Board of Radiology and the Residency Review Committee communicate are largely through the various Journals—The American Journal of Roentgenology, Radium Therapy & Nuclear Medicine; *Radiology; The Journal of the American Medical Association;* and the *Bulletin of the American College of Radiology*. There are a few letters or telephone calls from residents or preceptors to offices of the American Board of Radiology and the Council on Medical Education. It would seem that even this amount of communication is far from sufficient.

There is considerable question as to what the right method might be. After having numerous conversations with worried preceptors or teachers in the last few months, I am chagrined at how little I am able to communicate to them or how little they are able to absorb or remember, as indicated by the repetition of the same questions a few weeks to months later. It may well be that we all need a rather extensive course in communications. How can they be improved? Less than a month ago the American Board of Radiology at the invitation of the American College of Radiology tried an experiment at the Annual Meeting in New York concerning "The Thinking and Future Plans of the American Board of Radiology." Of great amazement to me was the small number of teachers, in comparison to the number of programs approved, who were present.

THE POSSIBLE NEW PROGRAMS

1. DIAGNOSTIC RADIOLOGY

It is understood that this will be a training program in diagnostic radiology, encompassing all aspects of roentgen diagnosis as well as the diagnostic applications of radioactive isotopes. It will require a 3 year period of training. It seems likely that only general hospitals having a large bed capacity and a wide variety of clinical material for training will be approved. A well balanced educational program at the resident level will require this large amount of diverse clinical material as well as continuous clinical teaching, and active investigative and research efforts. It will be expected that every resident will be active in some research assignment, and that the diagnostic applications of radioisotopes will be given proper emphasis. No affiliation from a parent institution will be counted as credit towards the 3 year program in diagnostic radiology.

2. THERAPEUTIC RADIOLOGY

This program likewise will require a 3 year residency including experience in the actual use of the accepted modalities of radiation therapy for all types and locations of cancer. It will also necessitate knowledge of basic sciences related to radiation therapy and malignant disease. General knowledge of techniques, methods

and results of other forms of cancer management must be studied so that the resident may be able to assess the merits of all methods of treatment of malignant disease. The institution offering such a program must have active services in cancer surgery and cancer chemotherapy as well as in radiation therapy, and again, no affiliation will be allowed. Staff supervision of the resident must be available and it is considered that at least one staff physician for each 3 residents in training must be available. A radiologic physicist must be a full-time member of the team. Patient material should consist not only of a large number but should be broad in scope, providing experience in all types of cancer amenable to radiation therapy. It is to be noted that the radiotherapist must have his own hospital service. Finally, research activities will be as equally emphasized as in the diagnostic program.

It is possible that each of us, you or I, could be enthusiastic or unhappy about these proposals. It is not expected that there will be unanimity of opinion, but it is believed that the provision of such programs will allow an experiment in radiologic education in the United States somewhat similar to the programs which have been carried out in other countries. It is my firm belief that there are few hospitals in the United States which can meet the requirements specified. In fact, I know that our own Department cannot, under present circumstances, meet the requirements either in diagnosis or in therapy.

FUTURE TRAINING PROGRAMS

Let us now leave the immediate factors which pertain to the present resident and consider some of the possibilities concerning future residency training programs.

A. There are those who suggest that the training programs of radiologists should be divided or, if general radiologic programs are retained, they should be at a very high and distinct level. These must be directed toward the person who is most interested in academic radiology and could be in the field of diagnostic radiology, therapeutic radiology, or radiology.

B. The alternative method would be the training program directed toward the education of the person who is interested in a future as a community radiologist, and in such a program the current type of combined training (radiology) might have strong possibilities. This would be especially true if divided programs became accepted widely for academic radiologists.

C. As another variant, there could be strict division of all training in radiology into diagnosis and therapy programs.

Personally, I am fully convinced that the best training program and the best future of Radiology rest in a program covering the entire field with opportunity for continued study in the selected branch at staff level and at "a living wage." I doubt that anyone can be a good radiation therapist without knowing the essentials of diagnosis. I doubt just as much that anyone can be a good general diagnostic radiologist without having the best basic background in radiation therapy. There are too many intertwining factors that are necessary in basic radiologic education as it stands at the moment. For instance, these might include radiologic (living) anatomy obtained only through diagnostic experience, and knowledge of tumors, including their natural history, found best in therapy practice. In spite of those who say that such dissection can be overcome, there are few persons who can complete a divided program and still have the necessary knowledge to be a true expert in the field without having seen both sides of the problem. Hopefully, it will be recognized that an intelligent and industrious person fulfilling the present day requirements—in the field of Radiology, Diagnostic Radiology or Therapeutic Radiology—is a very capable person and fully able to assume many responsible positions.

CONCLUSION

In approaching the conclusion, it might be said that the controversy as to whether

postgraduate education of the radiologist should be controlled by the Universities or by the Specialty Boards has not been entered. I have no intent to propose any alteration of the present program nor to be involved in this controversy. I will only say that the American Board of Radiology, which has had to work on its own without University support, has made remarkable accomplishments since 1934, and I am one who believes strongly that Radiology is now in its present position chiefly through the activities of this Specialty Board.

Preston Hickey would probably have had far better ideas to offer to you in 1962 concerning the education of the Radiologist and the Resident, but I am only able to leave you in somewhat of a dilemma because I have no immediate solutions. It is hoped that through conjoined thinking and planning all of us can improve the methods of training and the future of Radiology.

It has been an exceedingly great honor to meet with you tonight, to present these problems, to recognize abysmally that complete answers are not forthcoming at the moment, and to ask that you intensify your vigorous thinking so that the future of Radiology may continue to be as bright as it is at the present.

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ROENTGENOLOGIC MANIFESTATIONS OF JUVENILE RHEUMATOID ARTHRITIS*

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JUVENILE rheumatoid arthritis or Still's[51] disease is a systemic disease primarily affecting the connective tissue, particularly the joints. The pathologic process is basically the same as in adults,[14,41,42] but the clinical picture in children is frequently modified by severe constitutional and visceral reactions and disturbances in growth. While much has been written regarding the clinical aspects,[15,18,23,46] there have been relatively few reports primarily concerned with the roentgenologic manifestations.[39,45]

METHOD OF STUDY

Eighty patients were selected for study from a group of over 100 in whom rheumatoid arthritis began at or before age 14 and who were seen at the University Hospital since 1950. These cases were chosen on the basis of completeness of medical records and availability of roentgenograms. Many of these patients were referred and probably represented the more severe cases in any community. In all instances the diagnosis of rheumatoid arthritis was either "definite" or "classical" based on established criteria.[43] The criteria used for monoarticular rheumatoid arthritis were: (1) continuous, objective evidence of arthritis for at least 4 months, (2) compatible joint biopsy, (3) negative joint fluid culture, and (4) negative tuberculin skin test.

An effort was made to re-evaluate as many of these patients as possible, both clinically and roentgenologically. Thirty-five were currently being followed in the clinic and 22 others were recalled for examination, specifically for this study. Ten who were contacted by letter or telephone did not return. Four patients were dead. Follow-up information was not obtained in 9. The duration of clinical observation exceeded 10 years in 15 patients and 5 years in 26 others. None was followed for less than a year. Roentgenograms were available in childhood in all but 3 cases. At the time of this study 16 were known to have reached 20 years of age or more. As a result of this follow-up, roentgenologic re-evaluation of previously examined joints was possible, often after many years, regardless of the current disease activity.

CLINICAL DATA

The clinical data are summarized in Table 1. The female to male ratio was approximately 3 to 1. Seventy-seven were Caucasian and 3 were Negro. The onset of the disease was between 6 months and 14 years, with the mode at 3 years, and in one-third it occurred at or before the age of 3. Ten patients had either pericarditis, with or without myocarditis, or pleuritis. Splenomegaly was observed in 14 and lymphadenopathy was an outstanding manifestation in 7 at some time during the course of the disease. Uveitis or band keratopathy was occasionally noted.[50] Micrognathia was definite in 15 patients and possibly present in 7 others. The relative infrequency of subcutaneous rheumatoid nodules in children is well known.[11] Fifteen children developed an evanescent, distinctive rash of the erythema mutiforme type.[27] One or more serologic tests* for the rheumatoid factor were positive in 15 of the 69 patients tested. It is well known that such tests are

* Sensitized sheep cell hemagglutination, bentonite fixation and latex fixation tests. The number and type of tests employed varied.

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less frequently positive among children than adults.[38,59] The L.E. cell phenomenon was observed in 4 instances and while this test is most frequently positive in systemic lupus erythematosus, it is not diagnostic of that disease and has been reported in a significant number of cases of otherwise classical rheumatoid arthritis.[32] Chronic ulcerative colitis developed in 2 patients, 1 and 10 years, respectively, after the onset of arthritis. Two patients had psoriasis and in one this predated the arthritis by 5 years while in the other it followed 1 year later. The arthritis in these 4 patients was indistinguishable from that in the others.

Table II shows the distribution of arthritis at onset and over the course of the disease. Seventeen patients (21 per cent) had monoarticular disease and of these the knee was affected in 12. Other joints were subsequently involved in 11 of the 17 patients in a period ranging from 4 months to 5 years. The most frequently involved joints were the knee, ankle and wrist. Eight patients had the onset of symptoms in the cervical spine. The arthritis was predominantly asymmetrical in 20 per cent of the patients, excluding those in whom it remained monoarticular. While the hands were not commonly affected early, they tended to become involved as the disease progressed.

Table I

CLINICAL DATA—80 PATIENTS

Sex:	Female/Male	59/21
Age at Onset:	Less than 1 year	3
	1–3	24
	4–8	30
	9–12	14
	13–14	9
Pericarditis or pleuritis		10
Splenomegaly		14
Lymphadenopathy		7
Uveitis or band keratopathy		3
Micrognathia		15
Rheumatoid nodules		8
Skin lesions		15
Rheumatoid factor:		
No. positive		15
No. tested		69
Lupus erythematosus cell phenomenon		4
Steroid therapy		49
Chronic ulcerative colitis		2
Psoriasis		2

Table II

DISTRIBUTION OF ARTHRITIS—80 PATIENTS

	Per Cent		Per Cent
At Onset:*		During Course:	
Knee	36	Knee	90
Ankle	26	Ankle	70
Wrist	10	Wrist	70
Foot	10	Hand	55
Hand	10	Elbow	40
Cervical	10	Hip	35
Hip	5	Foot	35
		Shoulder	25
Monoarticular:	21	Cervical	21
		Sacroiliac	5
		Sternoclavicular	3

* In several patients more than one joint was affected simultaneously at onset.

ROENTGENOLOGIC MANIFESTATIONS

Destruction of joint cartilage and bone was usually a late manifestation, particularly in monoarticular arthritis and young children. Thirty-three patients showed no cartilage or bone erosion (Table III) and 10 of these had continuously active arthritis for

Table III

ROENTGEN FINDINGS—80 PATIENTS

	No. of Patients
No cartilage or bone destruction	33
Metaphyseal rarefaction	4
Soft tissue calcification	5
Periosteal bone apposition	18
Vertebral compression fractures	13
Epiphyseal compression fractures (severe)	9
Subluxations, extremities (excluding hands and feet)	12
Protrusio acetabuli	4
Ankylosis, extremities	15

at least 2 years; 3 of the 10 patients had monoarticular disease for 1, 2, and 6 years, respectively. The roentgenologic abnormalities frequently consisted simply of articular soft tissue swelling and regional rarefaction. Cartilage destruction finally occurred after 2 or more years in a number of patients, but bone erosion was generally a much later finding. This tendency toward cartilage and bone preservation has been noted previously.[41,42,45,51] However, the course of the arthritis was variable and joint destruction was occasionally early and progressive. The carpal ossification centers were completely resorbed in one patient within a 2 year period (Fig. 1, *A* and *B*), and in another, with widespread, severe arthritis, the carpal bones underwent erosion and partial resorption within a year. The diagnosis in the latter case was verified histologically following partial carpalectomy.

Band-like zones of metaphyseal rarefaction, identical to those seen in childhood leukemia,[5,40,49,52,57] were observed in 4 patients, aged 3, 6, 10 and 15 years, respectively. These transverse zones (Fig. 2*A*), measuring 2–12 mm. in width, occurred in the distal femur, proximal and distal tibia and fibula and in the distal radius—always in proximity to active arthritis. In one patient these persisted for 1 year and were associated with the development of "growth-arrest" lines of increased density which coexisted with the lucent zones (Fig. 2*B*). A poorly defined, localized area of metaphyseal rarefaction was seen in the distal radius of a fifth patient who had chronic arthritis of the adjacent wrist. Thin, translucent bands were present in the cervical vertebral bodies of 2 patients who had symptoms of cervical spondylitis (Fig. 3). These were closely parallel to the superior and inferior vertebral margins and *dense* transverse striations were evident in one of these patients 3 years later. These dense bands may have the same significance as "growth-arrest" lines in the peripheral bones.[47]

Periosteal bone apposition (Fig. 4), observed in 18 cases, was particularly common in the phalanges, metacarpals and metatarsals, adjacent to affected joints. It is frequently an early finding and may be due to inflammation of the joint capsule and subjacent periosteum. A nonspecific effect of chronic hyperemia may also be implicated, especially when the periosteum is extensively involved. The long bones were affected along their entire lengths in 2 patients with widespread, severe arthritis (Fig. 5). The prevalence of this finding in children and its relative rarity in adults are attributable to the ease with which the periosteum in the former is stimulated to form bone. Occasionally, the cortex of the involved bone remains thickened (Fig. 6), but more often it becomes thin and the marrow space widened, due to concomitant endosteal bone resorption. As a result, these bones, particularly the phalanges and metacarpals, appear rectangular in advanced cases.

Focal, soft tissue calcifications were noted in 4 patients, adjacent to involved joints—the knee in 2 and the wrist and hip, respectively, in 2 others. Their nature was uncertain although in 2 instances they were linear and might have been ligamentous. The other two may have represented calcified foci of muscular necrosis.[12] In one instance (Fig. 7) it could have been the result of previous joint fluid aspiration. To our knowledge, none of these patients had been treated with vitamin D.

Digital arterial calcification was an unexpected finding in one patient (Fig. 8, *A* and *B*), who exhibited arthritis of most joints and generalized skeletal demineralization associated with compression fractures of the vertebrae and peripheral epiphyses. She had been treated intensively with steroids for more than 2 years. Her blood urea nitrogen and serum calcium and phosphorus values were normal. Serologic tests for the rheumatoid factor were negative on at least three occasions. Forsyth[21] described a child with similar, severe rheumatoid arthritis who developed digital vascular calcification, which he considered

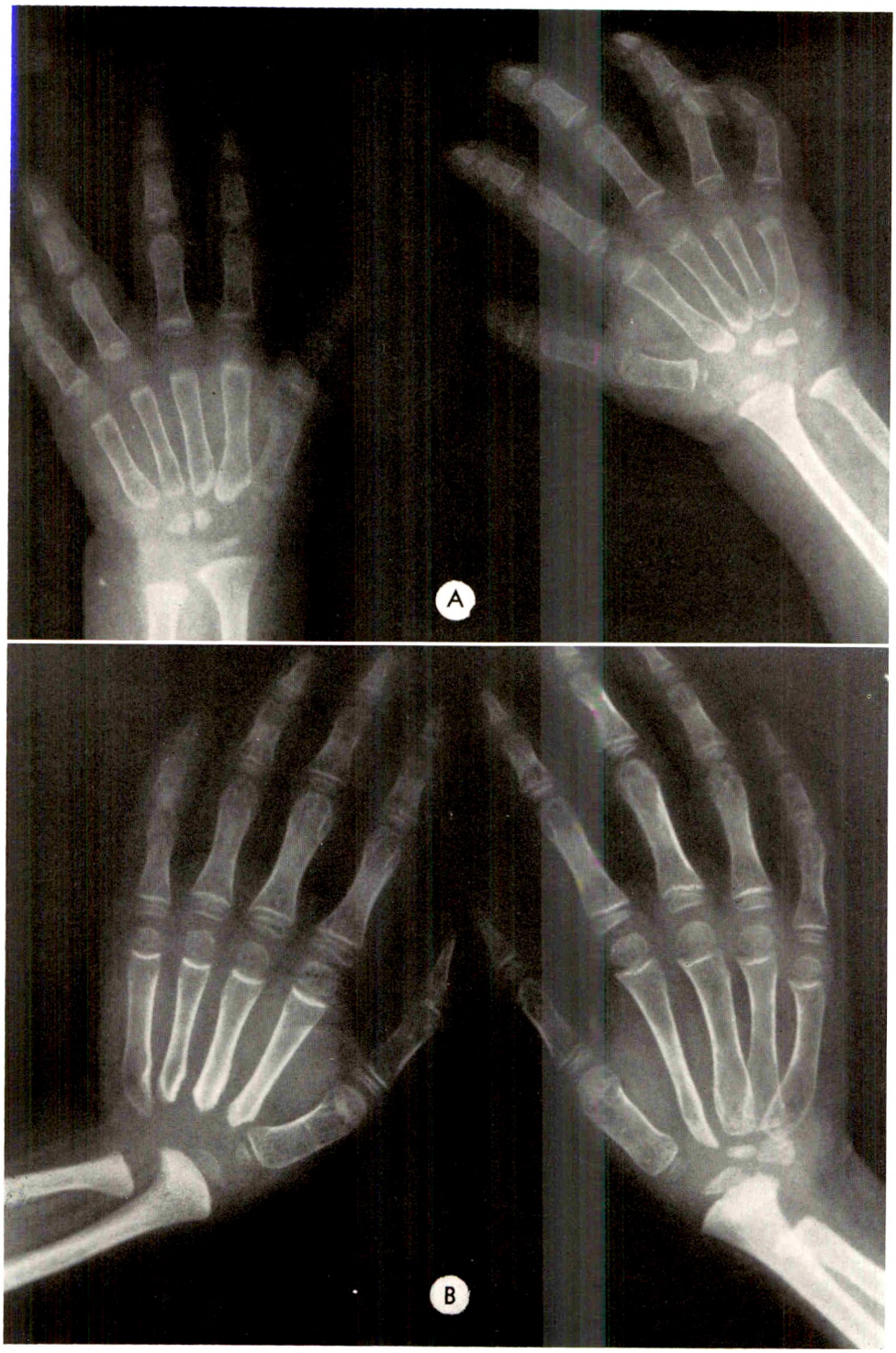


Fig. 1. (*A*) Aged 1. Rheumatoid arthritis began primarily in the wrists. There is minimal erosion at the bases of the metacarpal bones and slight flattening of the carpal ossification centers. (*B*) Aged 4. The carpal ossification centers are now completely resorbed on the left and compressed on the right. There is severe erosion of the bases of the metacarpals with "pencilling." Note the overgrowth of the radii with respect to the ulnae.

unique, and in whom amyloidosis was also present.

Compression fractures of the spine were observed in 13 patients, 12 of whom had received steroid therapy. These occurred principally in the dorsal region (Fig. 9) and were associated with generalized demineralization. One patient had a compression of L_5 and another showed flattening of the lower cervical vertebrae. These fractures predominated in the anterior portions of the vertebral bodies in one patient in whom there was a significant cervicodorsal kyphosis, secondary to extreme atlantoaxial dislocation, present for 4 years.

Compression fractures of the epiphyses of the extremities were frequently observed, especially in weight-bearing joints (Fig. 5) and in the small joints of the hands and feet (Fig. 8, *A* and *B*) where the effect of spasm

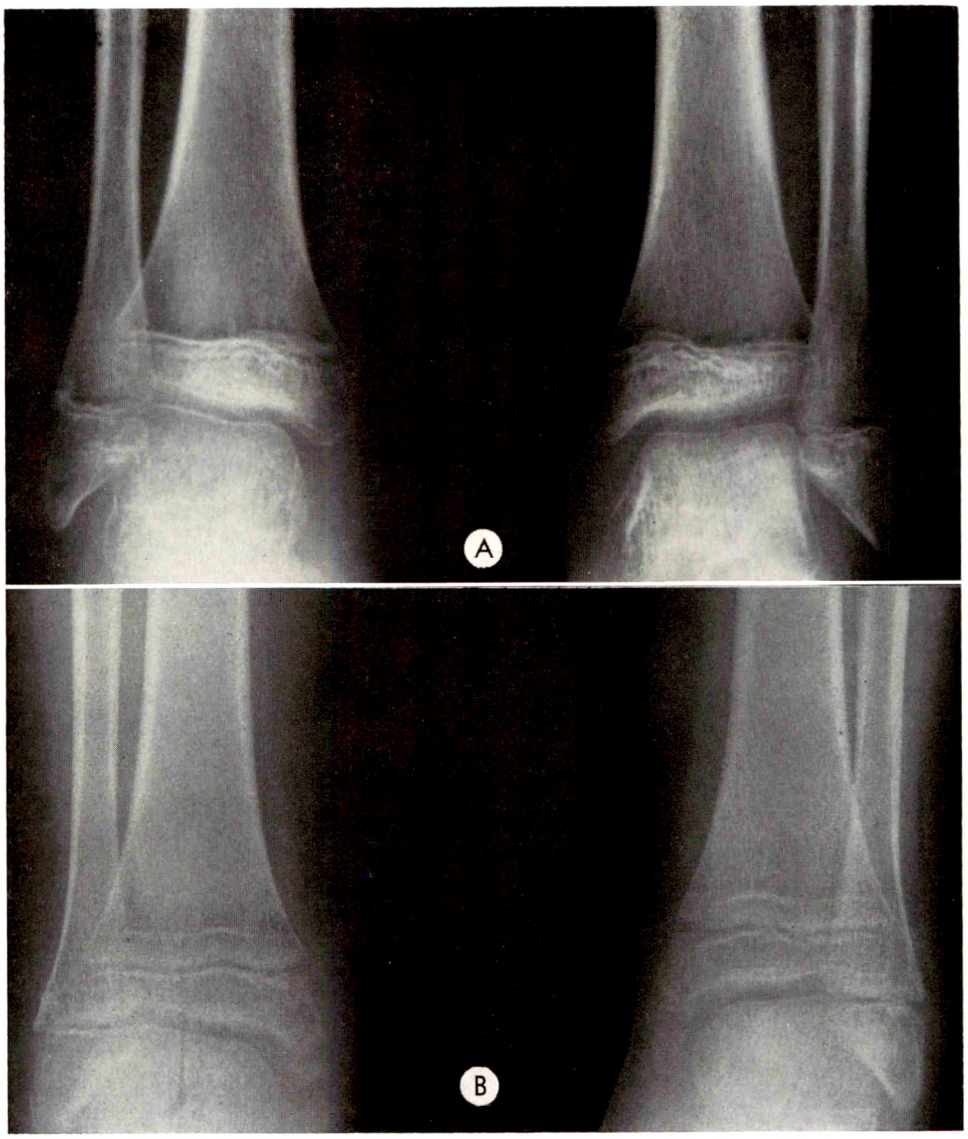


FIG. 2. (*A*) Aged 10. Rheumatoid arthritis of both ankles of one month's duration, associated with translucent metaphyseal bands. Similar lucent zones were evident at the knees. These are indistinguishable from those of childhood leukemia. (*B*) One year later. These bands of rarefaction are still present, though not as well defined, and are associated with transverse "growth-arrest" lines. A similar change was noted at the knees.



Fig. 3. Aged 11. Cervical pain began one year after the onset of arthritis in the peripheral joints at age of 10. Note the transverse bands of lucency adjacent to the superior and inferior vertebral body margins and the straightening of the spine. "Growth-arrest" lines were noted 3 years later.

and fibrosis of the intrinsic muscles, acting on demineralized bones, may be considerable. These were severe in 9 patients and lesser degrees of compression were observed in a number of others. Six of the 9 children also had vertebral compression fractures and spine roentgenograms were not available in 2 of the remaining 3. Seven patients had been treated with steroids. Flattening of the epiphyseal ossification centers of the metacarpals and metatarsals was often an early finding, at times associated with dense foci within the spongiosa due to trabecular compression (Fig. 10). A characteristic "cupping" of the ossification centers of the proximal phalanges, due to compression by the metacarpals or metatarsals, was frequent. The epiphyses of the remaining phalanges and distal radius were often

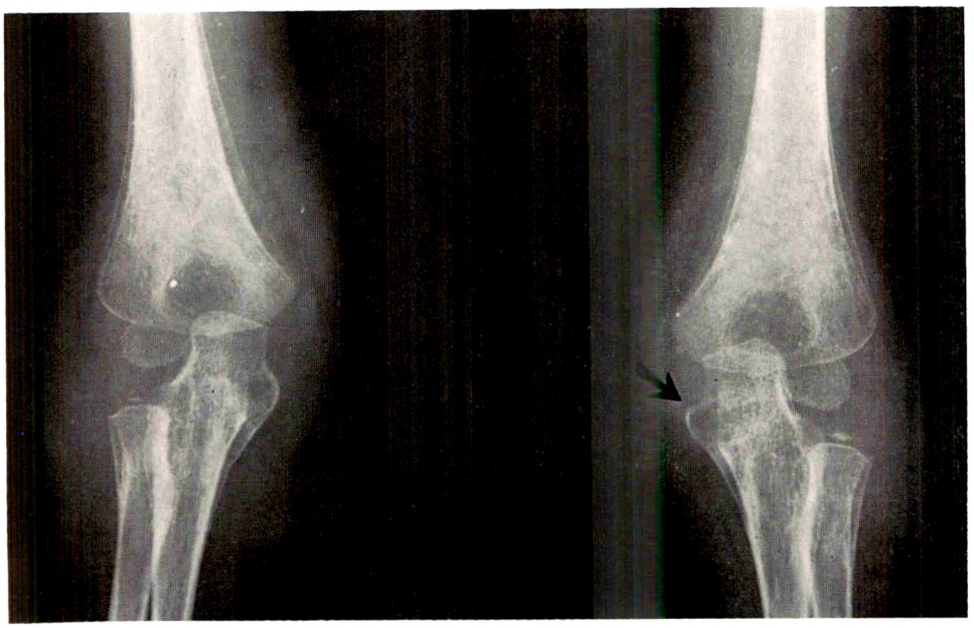


Fig. 4. Aged 4. Arthritis of the elbows of 1–2 years' duration associated with periosteal bone formation. Note the early erosion of the coronoid process on the left (arrow).

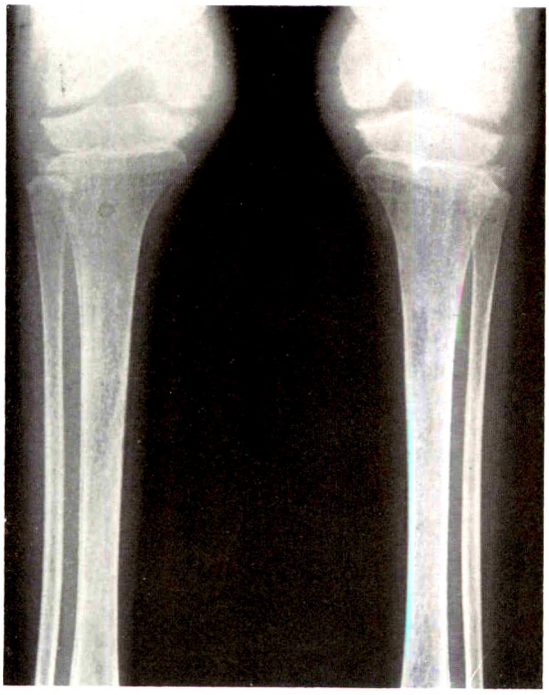


FIG. 5. Aged 6. Arthritis of the knees associated with destruction of cartilage and flattening of the tibial epiphyses due to compression fractures. No history of trauma. Note periosteal bone apposition along the tibial diaphyses.

flattened. Occasionally, rheumatoid destruction of the epiphyses with erosion and spiculation of the bone coexisted (Fig. 8*B*).

Subluxations of the joints of the extremities, other than those in the hands and feet, were observed in 12 patients and the hip was involved in 7 of these (Fig. 11). These subluxations often developed in the absence of significant bone erosion. An articular soft tissue mass was occasionally evident but it was felt that the cause was largely ligamentous destruction and muscular foreshortening due to fibrosis. Dislocation of the hip is frequent in this disease.[29,58]

Protrusio acetabuli in rheumatoid arth-

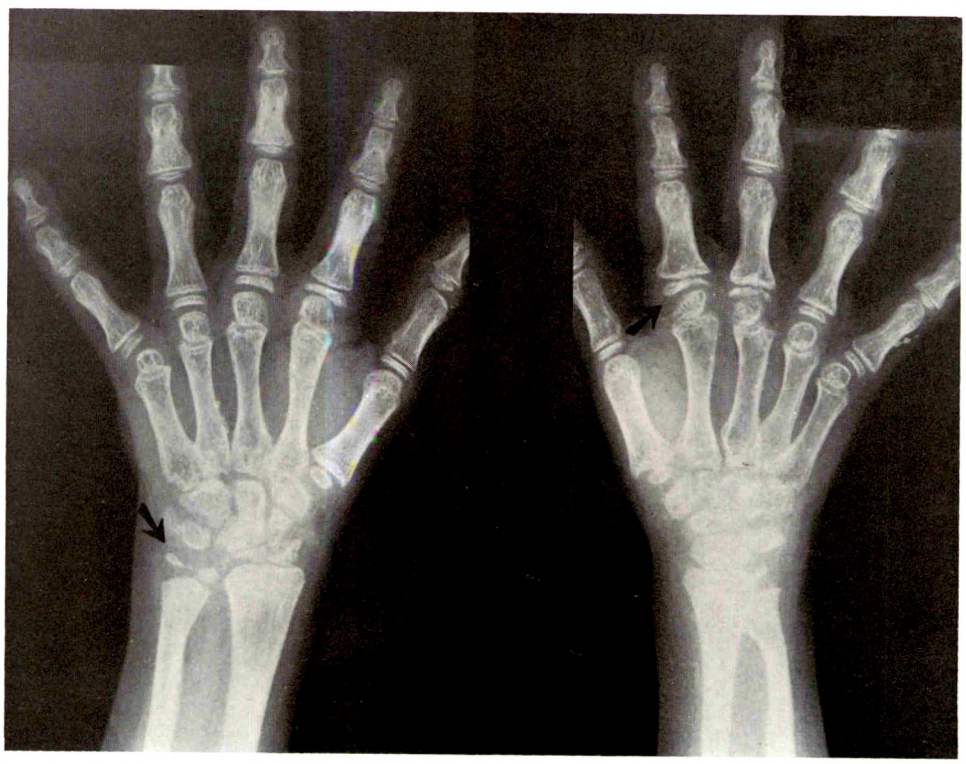


FIG. 6. Aged 7. Periosteal reaction in the phalanges and metacarpals causing cortical thickening. Note the brachydactyly and the epiphyseal compression on the right (arrow) and the separation of bipartite ulna ossification centers on the left (arrow). Observe the lucency in the metaphysis of the left radius and the disparity in the lengths of the right radius and ulna.

ritis may be observed both in adults[37] and children.[12,29,45] In this series it was mild in 3 cases and moderate in one other. Arthritis of the hip was present in all 4. It is probably due to acetabular demineralization, resulting from chronic inflammation and continued weight bearing.

Arthritis in the extremities resulted in *bony ankylosis* in 15 patients and of these, the wrist was affected in 13. Generally, at least one of the 3 groups of joints in the wrist (radiocarpal, intercarpal or carpometacarpal) remained unankylosed, allowing some motion. In one case wrist ankylosis developed within approximately 2 years after onset (Fig. 12). The tendency toward periosteal new bone formation may be responsible, at least in part, for the frequent development of bony ankylosis in these cases and immobility is undoubtedly an important factor.

Growth disturbances were difficult to quantitate but, as has been observed by others,[2] constituted one of the outstanding features of the disease. As in other chronic, systemic diseases, physical development was often retarded and transverse "growth-arrest" lines in the long bones were frequent. There was generalized retardation in epiphyseal maturation in one patient, but more commonly the epiphyses of affected joints showed an accelerated maturation and eventual overgrowth. Such enlarged epiphyses often presented a characteristic "ballooned appearance," with relative constriction just above and below the articular surfaces (Fig. 13). Their appearance suggested that the part of the epiphysis adjacent to the articular cartilage participated less in this hypertrophy than the remainder, nearer the hyperemic joint capsule. A frequent manifestation in an affected extremity was generalized demineralization and reduction in the long bone diameters and soft tissues. The entire lower extremity and pelvis on the ipsilateral side were occasionally so affected, though the arthritis was confined to the knee. This nonspecific atrophy may be observed in other diseases.[55] At times such atrophy was accompanied by overgrowth in the length of the long bones and, occasionally, adjacent bones were affected unequally, causing bowing deformities. At times all the long bones appeared generally short. The bones of the hands and feet often appeared uniformly small (brachydactyly) without premature epiphyseal fusion (Fig. 6). Some patients exhibited undergrowth of one or two such bones in which premature epiphyseal fusion was evident. One such patient showed symmetrical, short fourth metatarsals and phalanges with epiphyseal fusion but without clinical or roentgenologic evidence of arthritis in the joints of this digit (Fig. 14). However, this patient did have arthritis of multiple joints, including the ankles. In one patient with chronic arthritis of both knees but without bone destruction, only the medial portions of the proximal tibial and distal femoral epiphyses became fused (Fig. 15).



FIG. 7. Arthritis of 2 years' duration, associated with juxtaarticular calcification (arrow).

The temporomandibular joints were examined in 11 of the 15 patients with *micrognathia* and laminagraphy was employed in 8. These examinations were undertaken largely for the purpose of this study rather than for specific complaints referable to these joints. A history suggesting temporomandibular arthritis was conspicuously absent in most cases. The body and vertical rami of the mandible appeared short and the mandibular notches were often wide. Characteristically, the fossae were broad and shallow and the condyles were poorly differentiated (Fig. 16 and 17). These growth disturbances appeared out of proportion to the functional and morphologic

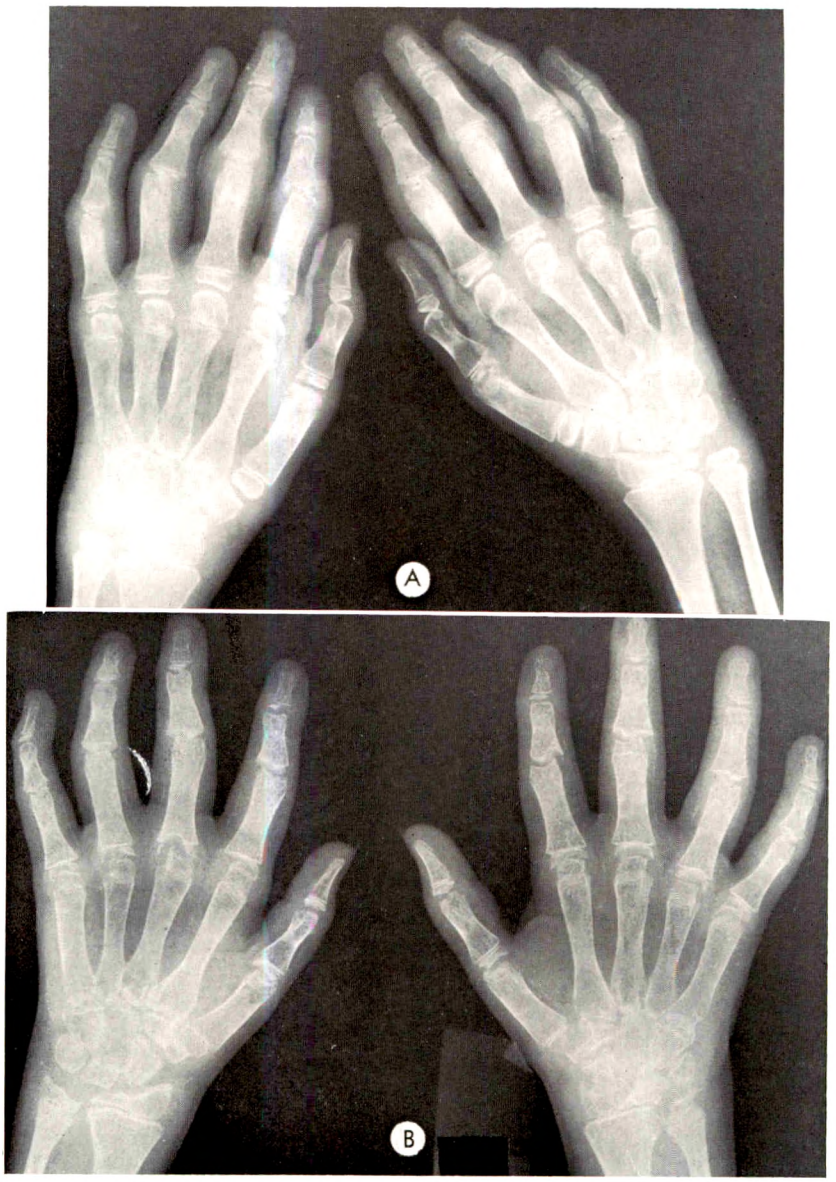


FIG. 8. (*A*) Aged 8. Severe arthritis and demineralization with epiphyseal compression and soft tissue swelling about the proximal interphalangeal joints. Note periosteal bone formation of the metacarpals. (*B*) One year later. Epiphyseal "cupping" due to compression with superimposed erosion and spiculation of the bone. Note the flattening of the radial and ulnar epiphyses and the development of digital arterial calcification bilaterally.

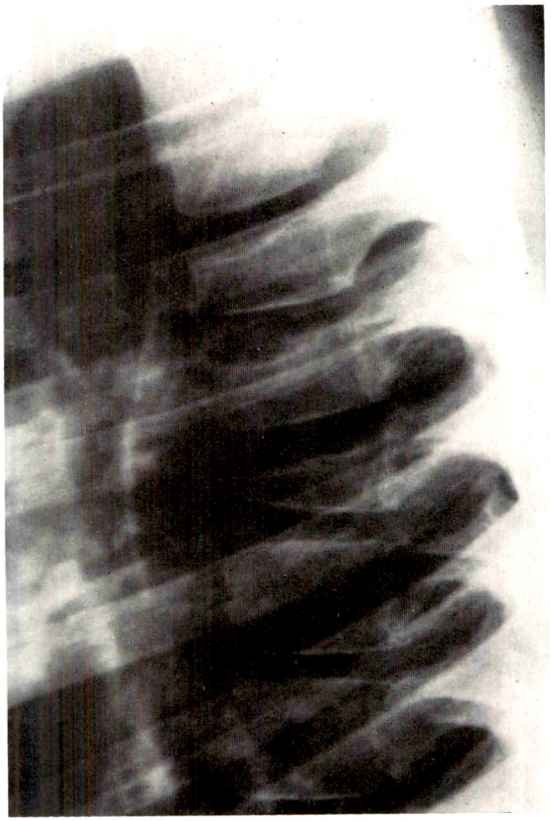


Fig. 9. Same case as in Figure 8. Compression fractures of dorsal vertebrae during steroid therapy. Note that the heights of the intervertebral disks are not reduced.



Fig. 10. Aged 6. Same case as in Figure 5. Fulminating arthritis of 3 years' duration with severe demineralization and early "cupping" of the epiphyses of the proximal phalanges. Note the dense foci due to trabecular compression in the metacarpal epiphyses (arrow). Observe the thin cortices and rectangular shape of the metacarpals.

changes in the joints themselves and were always bilateral and, for the most part, quite symmetrical. Evidence of cartilage and bone erosion was observed in 2, possibly 3 cases (Fig. 18), one of whom also had changes of secondary osteoarthritis. The latter was conspicuously absent in 3 others with severe micrognathia who were examined in adulthood. While joint motion was in most cases abnormal, the degree of excursion was often surprisingly good.

Spondylitis was frequent (Table IV) but its appearance differed from "ankylosing spondylitis."* Cervical spine roentgenograms were obtained in 28 patients, most of whom had neck symptoms, and in 17 there was roentgenologic evidence of cer-



Fig. 11. Aged 11. Arthritis of the hips for 9 years. There is destruction in both joints with a shelf-like proliferation of bone of the left acetabulum secondary to subluxation. No previous hip surgery.

* This term is used here synonymously with rheumatoid spondylitis, Strümpell-Marie-Bechterew disease, pelvospondylitis ossificans and spondylose rhizomelique.



FIG. 12. Aged 12. Extensive ankylosis of the intercarpal and carpometacarpal joints. Ankylosis was appreciable within 2 years after the onset of arthritis in the wrists. Note that the joints of the hands are spared.

vical spondylitis. Eleven had vertebral subluxation and in 9 this occurred only at the atlanto-axial joints* (Fig. 19, *A–C*). Five of these 9 patients were between 5 and 10 years of age and 4 were over 17 at the earliest date that the dislocation was noted. Apophyseal joint ankylosis predominated in the upper half of the cervical spine, par-

* While the normal upper limit for the atlanto-odontoid interval has been studied in adults,[25,28] the normal range of this measurement in children at various ages is unknown. For this study an atlanto-odontoid interval of 5 mm. or greater was arbitrarily chosen as definitely abnormal.



FIG. 13. Aged 42. Chronic arthritis of the knees since early childhood. There is overgrowth of the epiphyses presenting a characteristic "ballooned appearance" with relative constriction just above and below the articular surfaces. Note secondary osteoarthritic changes.



Fig. 14. Aged 13. Premature fusion of the epiphyses of the fourth digit bilaterally with selective demineralization. The adjacent joints appear normal and there was no history of arthritis of the joints of the feet though the ankles were affected. Roentgenograms at the age of 2 showed normal metatarsals.

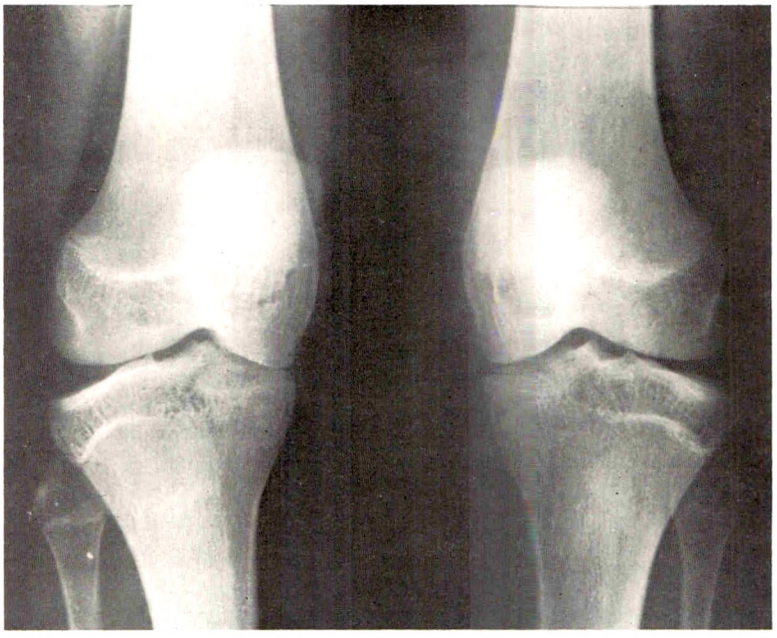


Fig. 15. Aged 14. Chronic arthritis of both knees of several years' duration with premature fusion developing in the medial portions of the epiphyses with a resultant deformity. Earlier roentgenograms showed normal epiphyses.

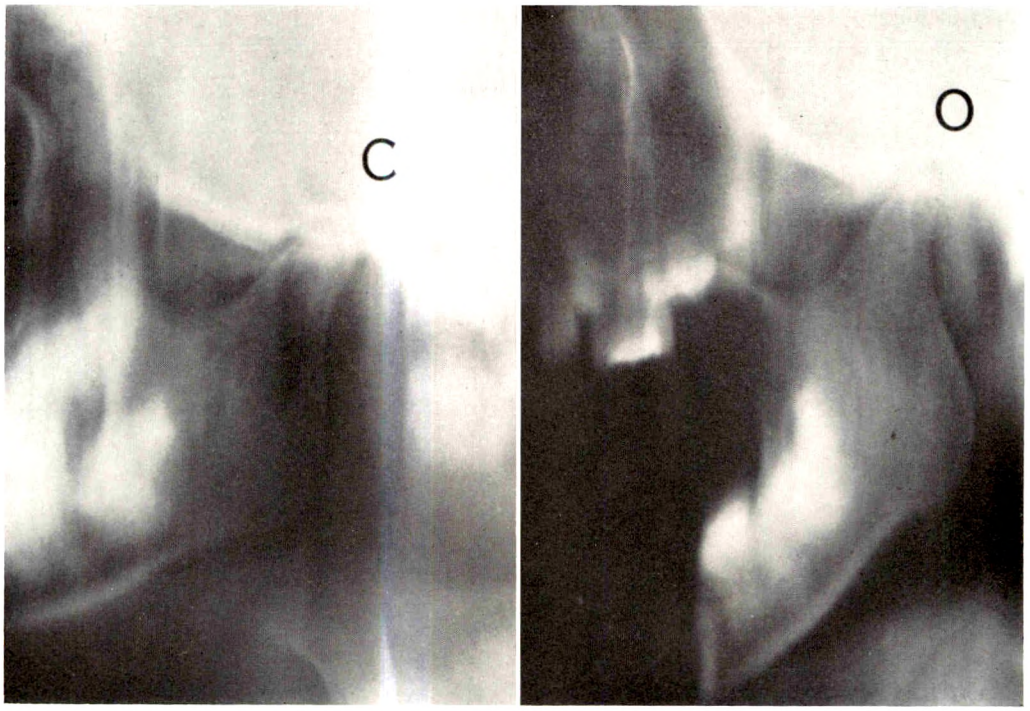


FIG. 16. Aged 11. Temporomandibular joint laminagrams. The condyle is not well differentiated and the fossa is somewhat shallow. There are no bone erosions but the cartilage space appears slightly diminished and excursion is clearly limited (C, closed mouth; O, open mouth). The opposite joint had the same appearance.

ticularly at C_2–C_3 (as seen in the lateral projection) and was observed in 11 individuals. There were no patients in whom the lower cervical segments were ankylosed and the upper ones spared. Paraspinal ossifications were conspicuously absent and only 2 of the patients with cervical spondylitis had sacroiliac arthritis. One 7 year old exhibited periosteal bone proliferation of the odontoid process on laminagrams, in association with chronic, nontraumatic, atlanto-axial dislocation (Fig. 20). In a 42 year old patient who had apparently sustained a similar dislocation, the odontoid was replaced by a block of bone which was fused to the atlas and basiocciput (Fig. 21). Growth disturbances in the cervical spine were frequent. The vertebral bodies were reduced in both height and anteroposterior diameters, particularly at the levels of apophyseal ankylosis (Fig. 19, *A–C;* 21; and 22, *A* and *B*). The intervertebral disks were diminished in height and at times were almost nonexistent (Fig. 22*B*). Occasionally, disk calcification was evident in adulthood (Fig. 21). Similar atrophic changes of the vertebrae and disks were observed in the dorsolumbar spine in 4 patients, suggesting that the adjacent apophyseal joints might have been involved (Fig. 23, *A* and *B*). Three of these patients were followed beyond the age of 20 and, although the cervical spine was affected in

TABLE IV

SPONDYLITIS

	No. of Cases
Cervical spondylitis	17
with subluxation	11
with apophyseal ankylosis	11
with sacroiliac arthritis	2
with paraspinal ossification	0
(?) Dorsolumbar involvement	4
Sacroiliac arthritis without spondylitis	2

all 4, the sacroiliac joints remained normal and paraspinal ossifications did not develop. There were 2 patients with sacroiliac, as well as extraspinal arthritis but without other evidence of spondylitis; it was not clear from the roentgen findings whether these represented cases of ankylosing spondylitis in which spinal changes had not yet developed, or rheumatoid arthritis.

Secondary osteoarthritis was quite common, even in childhood and adolescence, and is to be expected as a sequel to severe arthritis, particularly in active and weight-bearing joints. At times proliferative changes were marked, apparently in response to abnormal mechanical stress, associated with major joint deformity (Fig. 11 and 24).

DISCUSSION

The distribution of arthritis in these patients corresponds with that reported in other series,[15,18,23,45,51] and differs notably from the adult disease in its predilection for parts other than the hands and feet, particularly at onset, and in the greater prevalence of monoarticular disease. Sairanen[45] states that the fingers are more commonly involved at onset if the disease begins beyond the age of 10 years. Symptoms of cervical spondylitis may occur early, prior to involvement of the peripheral joints.[12,33,54] The childhood distribution of arthritis also differs from the pattern in ankylosing spondylitis where it is the hips and shoulders that are the most common extraspinal joints affected and the sacroiliac

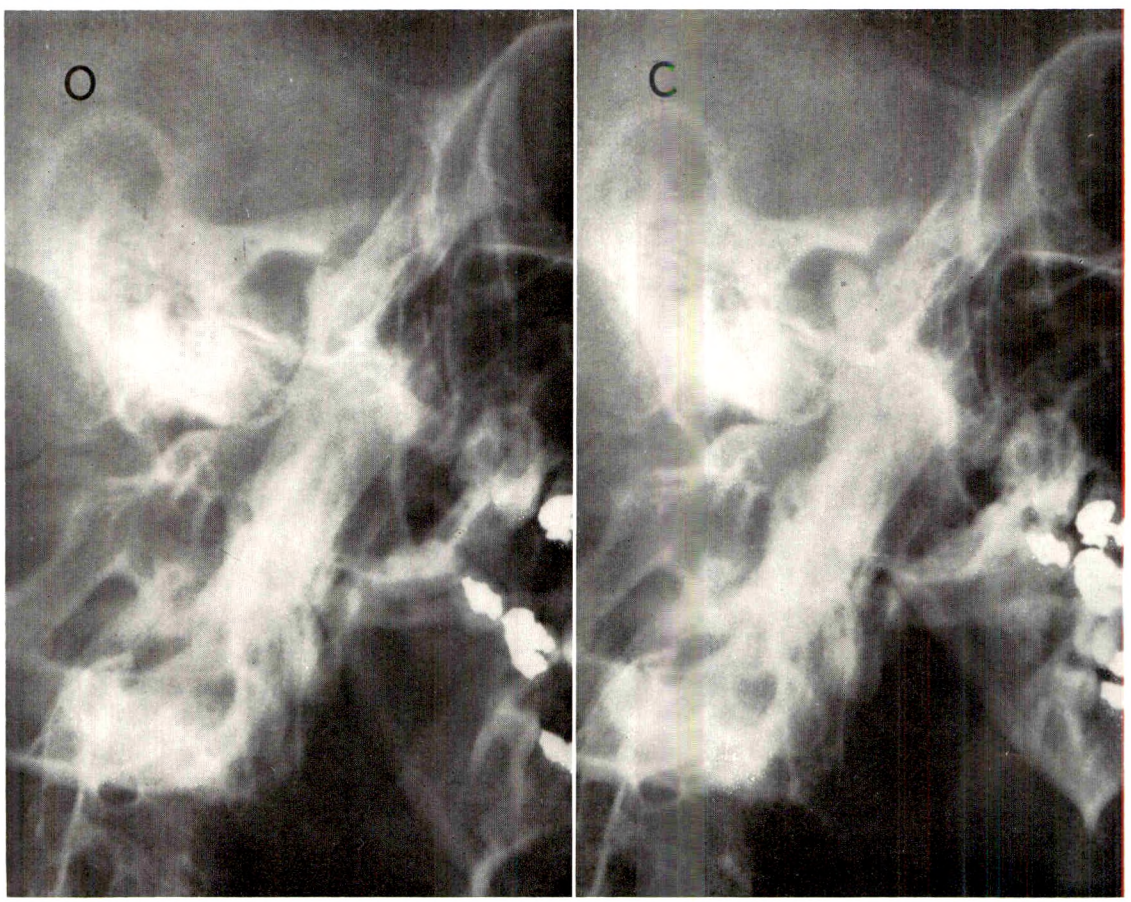


Fig. 17. Aged 20. Temporomandibular joint. The fossa is broad and shallow and the vertical ramus is short with a small condyle. Excursion is good, though not normal, and there are no erosions or proliferative changes (O, open mouth; C, closed mouth). There was no history of arthritis of these joints.

joints are almost always involved on onset. It is of interest that in children the parts most frequently affected, the knee, ankle and wrist, correspond with the sites of most active bone growth. These are also the sites most apt to show roentgenologic changes in other childhood diseases affecting the skeleton such as scurvy, rickets and leukemia.

Band-like zones of metaphyseal rarefaction constitute the most common roentgenologic finding in childhood leukemia but are nonspecific and may be observed in other conditons.[5,40,49,56,57] Its occasional occurrence in juvenile rheumatoid arthritis is significant and should be emphasized since childhood leukemia may mimic this disease clinically. LoPresti[34] mentions this finding in a 15 month old child with rheumatoid arthritis. Willson[57] reviewed 25 cases of juvenile rheumatoid arthritis and sought for, but was unable to detect, such lucent zones. He states that above the age of 2 years these transverse lucent lines are rarely seen in any other disease besides leukemia and therefore take on a certain specificity beyond this age. Its occurrence in 4 patients in this series suggests that this viewpoint should be accepted with reservation. These radiolucent zones are postulated to represent a depression in endochondral bone formation related to severe systemic illness.[30,49,52] They can spontaneously disappear in leukemia in remission[49] and may be replaced by "growth-arrest" lines.[40] Jaffe[30] states that an additional factor in leukemia may be the resorptive pressure effect of leukemic tumor tissue in the marrow upon adjacent trabeculae. Likewise, in rheumatoid arthritis a disturbance in endochondral bone formation may not be the sole factor, for these lucent bands may also develop in adolescents with this disease whose epiphyses have recently fused. Inasmuch as the metaphyses are particularly hypervascular in the young, especially in the presence of chronic inflammation, and since this area is composed largely of trabecular bone, these zones may represent trabecular atrophy due to persistent hyperemia. Their occurrence in the cervical spine in patients with symptoms referable to this region presumably has the same pathologic basis for there is a rich network of vessels on the vertebral side of the cartilaginous plates. The later develop-

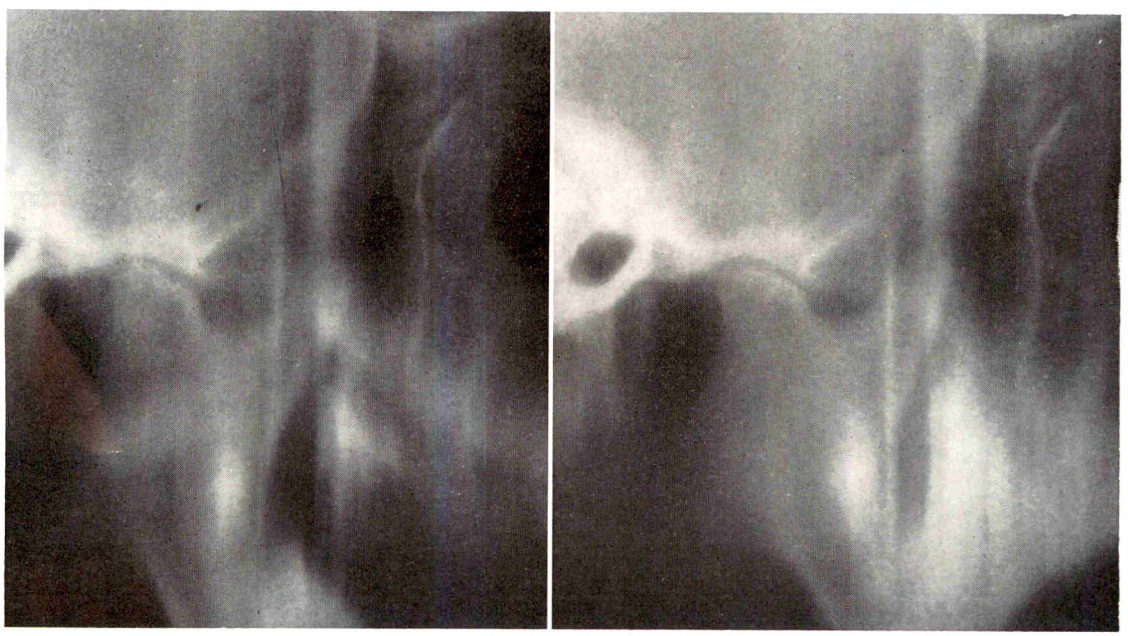


FIG. 18. Aged 10. Rheumatoid arthritis began at age of 4. The cartilage space is reduced and there is erosion of bone. A condyle is not evident, the fossa is shallow and the ramus is short. Joint motion is quite limited.

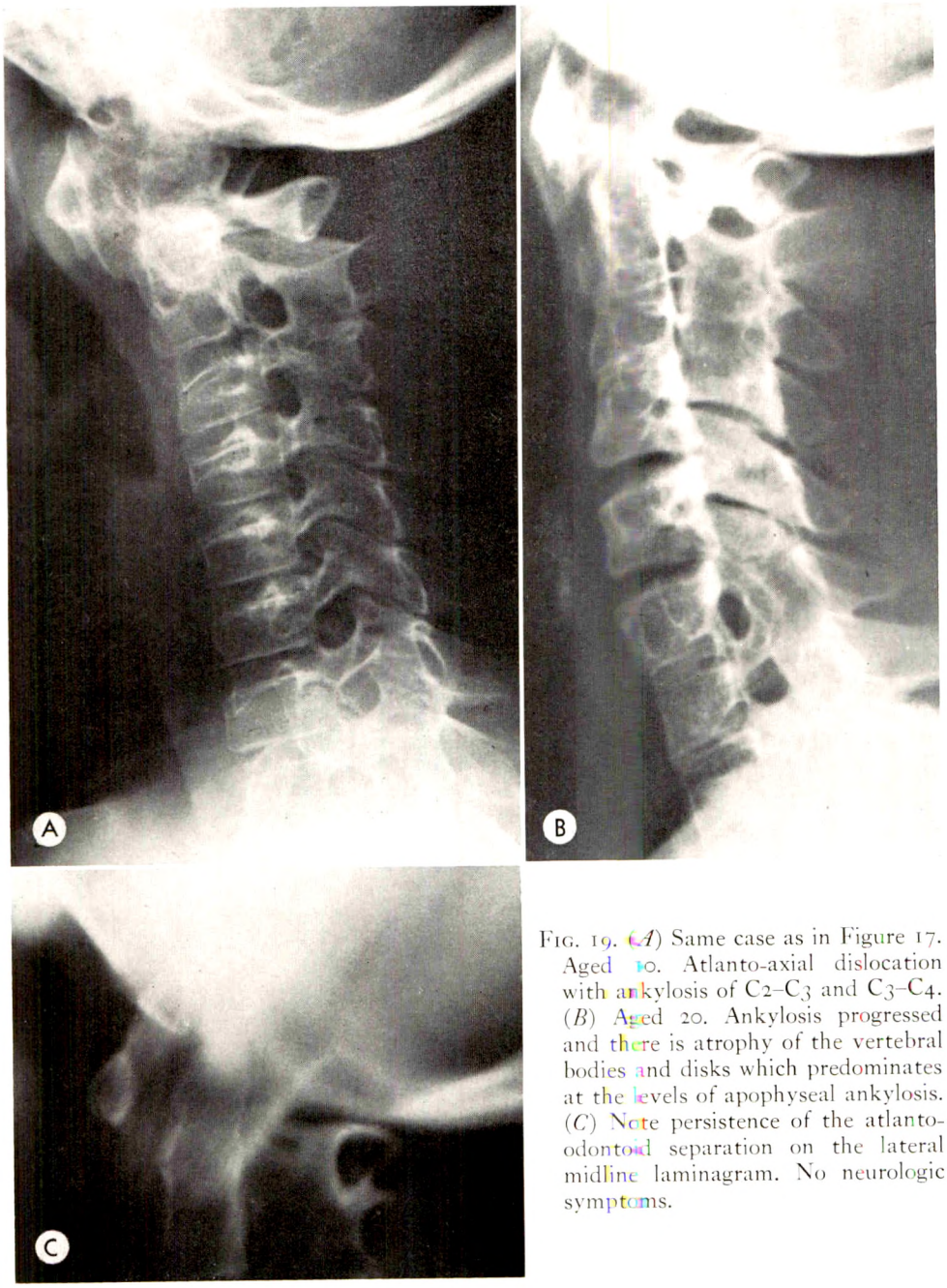


Fig. 19. (*A*) Same case as in Figure 17. Aged 10. Atlanto-axial dislocation with ankylosis of C_2–C_3 and C_3–C_4. (*B*) Aged 20. Ankylosis progressed and there is atrophy of the vertebral bodies and disks which predominates at the levels of apophyseal ankylosis. (*C*) Note persistence of the atlanto-odontoid separation on the lateral midline laminagram. No neurologic symptoms.

ment of "growth-arrest" lines in these vertebrae seems to support the concept that it is the same phenomenon as in the long bones. In one patient there was a suggestion of similar lucent zones in the dorsal spine (Fig. 23*A*).

Vertebral compression fractures have been described in this disease,[3] particularly in steroid-treated patients, but surprisingly little attention has been paid to the compression fractures of the epiphyses of the extremities. Pickard[41] observed flattening of the lateral femoral condyle in one patient at surgery though the articular cartilage was not eroded. He noted that "demineralization of the epiphyses may result in in-

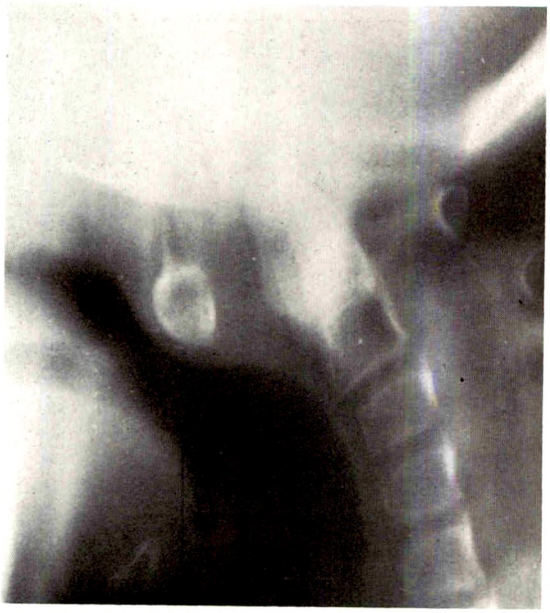


FIG. 20. Same case as in Figure 18. Aged 8. Lateral midline laminagram. Atlanto-axial subluxation developed at age of 4, one year after the onset of peripheral arthritis. There is widening of the odontoid due to periosteal bone apposition. No neurologic symptoms.

creased fragility . . . crumbling and early fusion may ensue." The mechanical effect of muscular foreshortening and weight bearing on the demineralized epiphyses may play a significant role in growth inhibition. In 2 adults with severe arthritis since childhood, there were central, notch-like defects at the ends of the phalanges and metacarpals (Fig. 24).

Growth disturbances, adjacent to involved joints, are often characteristic and constitute a hallmark of the disease in children. The factors involved appear to be independent. Thus, an increase in epiphyseal size is frequent but is not necessarily concomitant with overgrowth in the length of the long bone and is, furthermore, often associated with reduction in its diaphyseal diameter. Unilateral lengthening of a long bone may be observed in other conditions[17] and there is evidence to suggest that chronic hyperemia may be the common denominator.[9,21,31] Accelerated epiphyseal maturation and eventual overgrowth has also been attributed to this factor. With regard to growth inhibition, there is undoubtedly a generalized effect due to the chronic, systemic nature of the disease[2] and prolonged steroid therapy may play a role.[6,7] Growth of the long bones may be so severely inhibited as to mimic the chondrodystrophies.[10] Apparently, there is also a local effect, the mechanism of which remains obscure. Arthritis began in one child at the age of 2 and was confined to the left wrist and both knees (Fig. 25*A*) where it remained for one year after which the disease cleared completely and left no joint deformity or limitation. Despite this short duration and benign course, roentgenograms at age 13 showed that the carpal bones on the left were notably smaller than on the normal right side (Fig. 25*B*). Both this case and the one

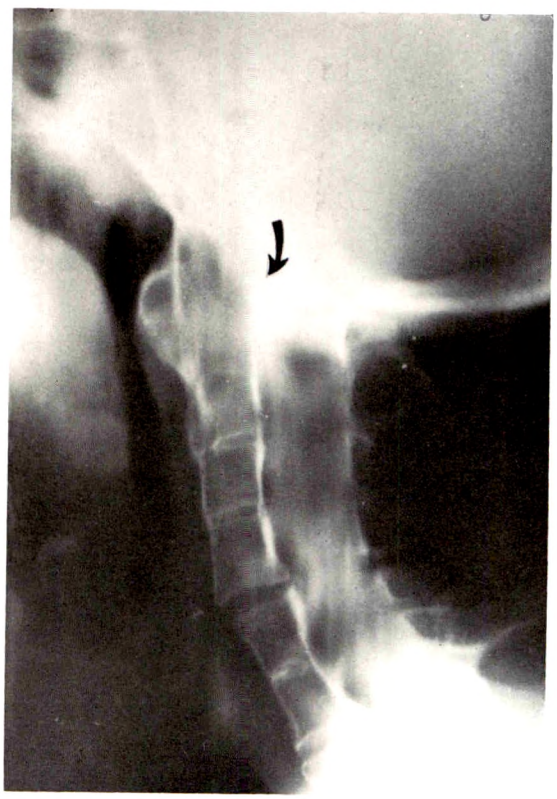


FIG. 21. Aged 42. Onset of disease at age one. The odontoid is widened (arrow marks most posterior margin) and fused to the atlas and basilar process. The appearance indicates previous anterior subluxation of C1 with subsequent atlanto-odontoid ankylosis. This is probably the appearance that will ultimately be assumed by the patient shown in Figure 20. Note the size and shape of the vertebral bodies and disks and calcification in the latter.

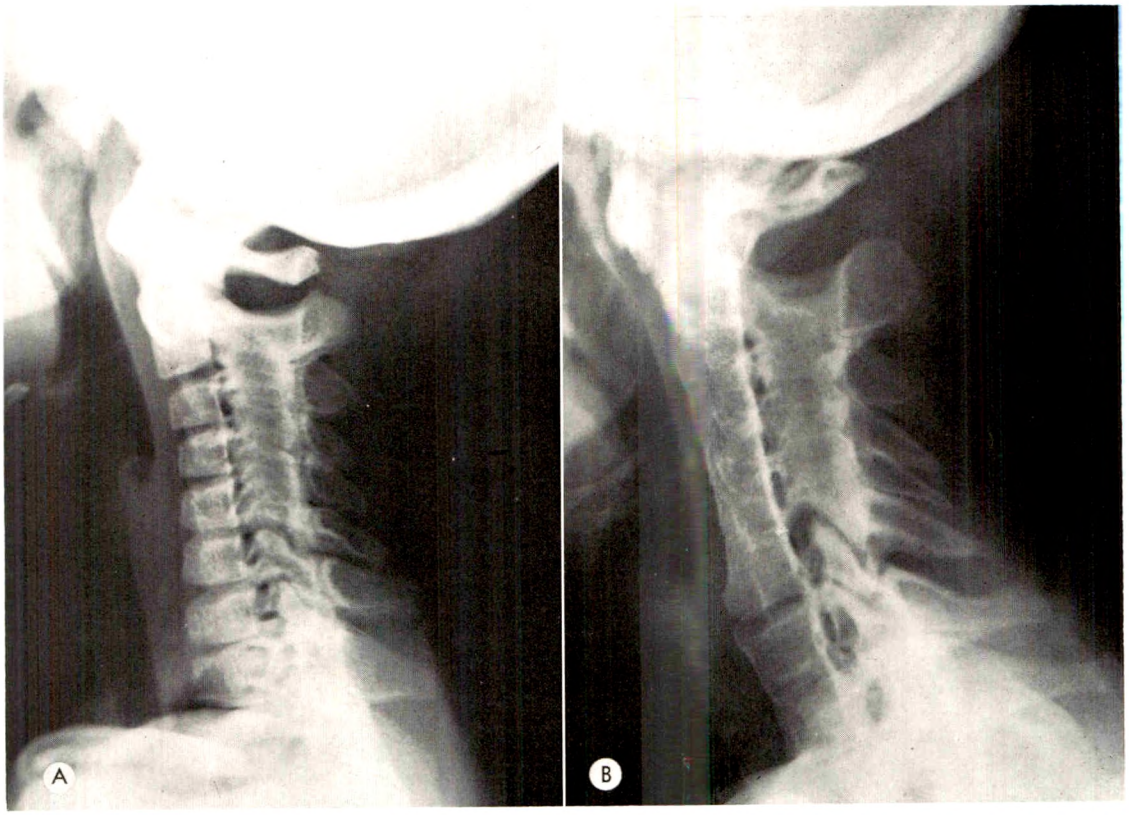


Fig. 22. (*A*) Aged 7. (*B*) Ten years later. A severe effect on vertebral growth with striking atrophy of the intervertebral disks. Note pointing of the spines and slight atlanto-odontoid separation.

illustrated in Figure 14 suggest that growth inhibition, while apparently a local effect, may not depend on the arthritis *per se*. Attempts have been made to relate the type of atrophy seen in the cervical vertebrae and long bones to nonuse or neurogenic factors and there are arguments in favor of both theories.[55] Brachydactyly is quite common in juvenile rheumatoid arthritis and has been attributed to premature epiphyseal fusion.[39] However, this may be an indication of the cessation of bone growth rather than its cause. It is apparently quite difficult to inhibit epiphyseal growth by mechanical trauma,[22,26] and in some cases of premature fusion there is little, if any, bone destruction (Fig. 14 and 15). Furthermore, many of our patients with generalized brachydactyly showed no such epiphyseal fusion. Finally, there is an important local effect due to the disease itself. Children's bones grow and model as a response to mechanical requirements.[53] Joint destruction and subluxation, inflammation of periarticular connective tissue and muscular spasm and fibrosis, with eventual contracture, all produce abnormal mechanical stresses which are bound to lead to modeling deformities and stunted, uncoordinated and sometimes bizarre growth.

Inhibition of mandibular growth in this disease is well known,[2,12,20,45,51] particularly if the onset is early in childhood. The length of the mandible and the height of its ramus are determined almost exclusively by its condylar growth center. Unlike the epiphyses of long bones, growth of the condylar cartilage, in which bone arises, is derived by apposition from a layer of precartilaginous connective tissue which lies beneath the fibrous articular surface[9,44,53] (Fig. 26). Since the fibrous layer lines the joint cavity, there is no cartilaginous barrier to protect the precartilaginous zone. This delicate construction suggests that

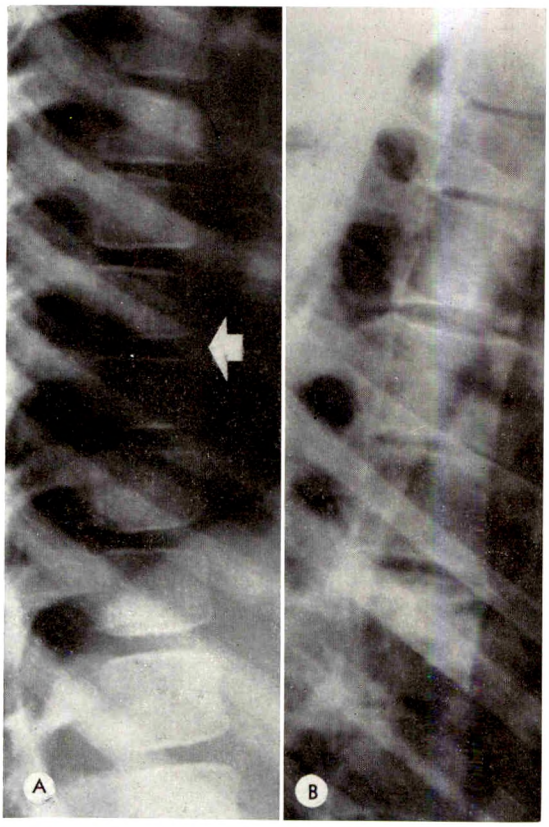


FIG. 23. (*A*) Aged 7. Minimal reduction in the height of one of the dorsal intervertebral disks (arrow). Note suggestion of transverse lucent bands. (*B*) Ten years later. Several disks are reduced in height and the vertebral bodies show variation in size and shape. The adjacent apophyseal joints were difficult to evaluate. Paraspinal ossifications were absent and the sacroiliac joints were normal.

there may be a particular susceptibility to injury, especially in early life.

There is little doubt that rheumatoid arthritis may affect the temporomandibular joints in children as well as adults. This was obvious roentgenologically in 2 cases with definite bone erosion. In another patient there was a dramatic disparity between the size of the condyles and fossae. The former were symmetrically tiny but the fossae were of normal size and shape (Fig. 27, *A* and *B*). If the development of the fossa is determined by its functional relation to the condyle,[48] then this appearance suggests that the condyle was altered after normal growth occurred. However, whether the change was entirely due to rheumatoid erosion is uncertain. It is of interest that this patient never had symptoms of temporomandibular arthritis. This was true of a number of others with severe micrognathia and was also noted by Sairanen.[45] It need not, however, negate the possibility of previous arthritis of these joints since rheumatoid synovitis in children is often painless.[23] It is reasonable to suggest, in view of the frequent disparity between the degree of growth retardation



FIG. 24. Same case as in Figure 21. Aged 35. Notching of epiphyseal ends of the phalanges (arrow) and metacarpals and secondary degenerative changes. Note the bizarre deformity of the long bones and carpal subluxations.

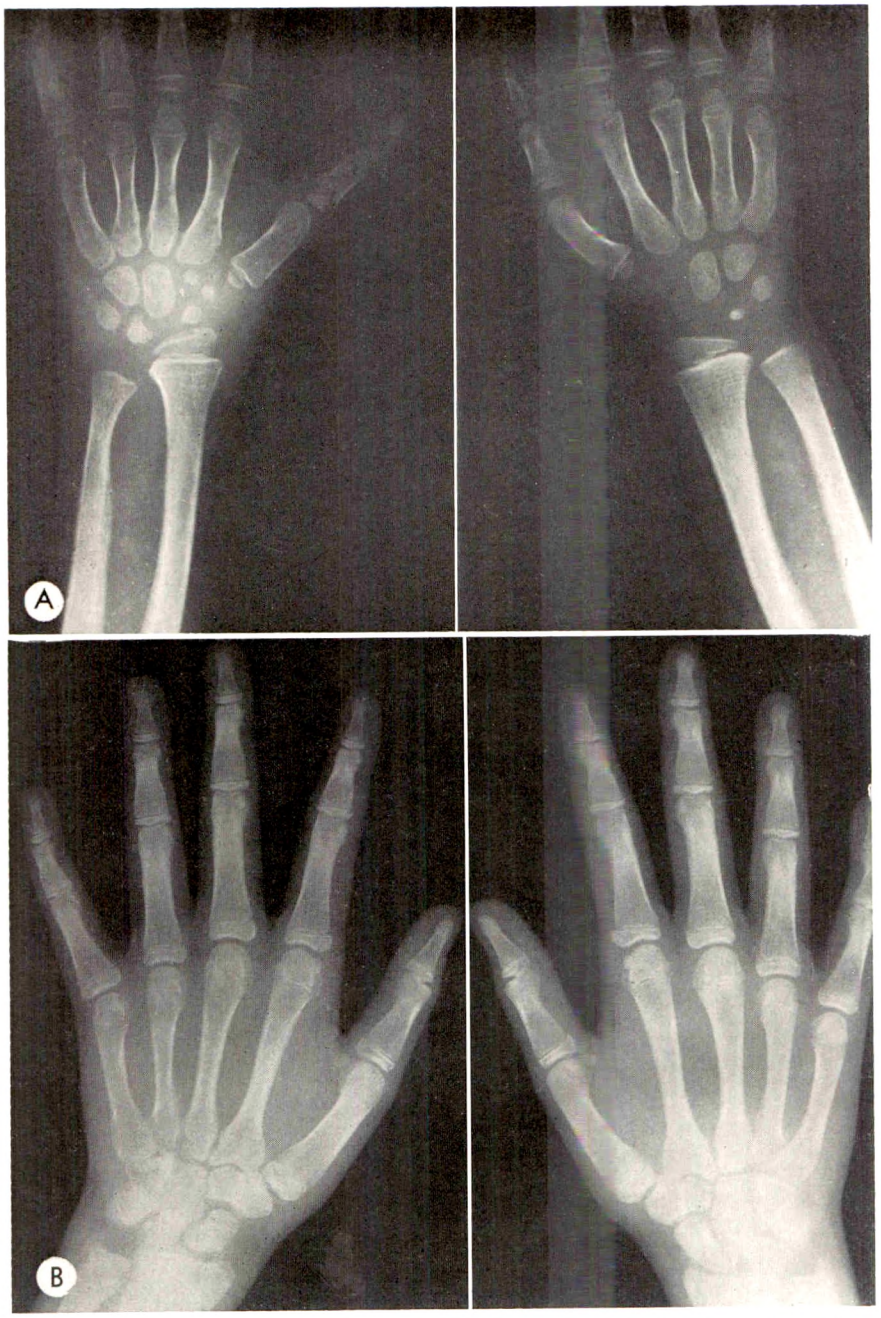


Fig. 25. (*A*) Arthritis, with onset at age of 2, was confined largely to the left wrist and both knees and lasted one year. Note the soft tissue swelling and accelerated epiphyseal maturation on the left. (*B*) Aged 12. Despite the short duration and benign course of the disease, the carpal bones on the left are now smaller than those on the right.

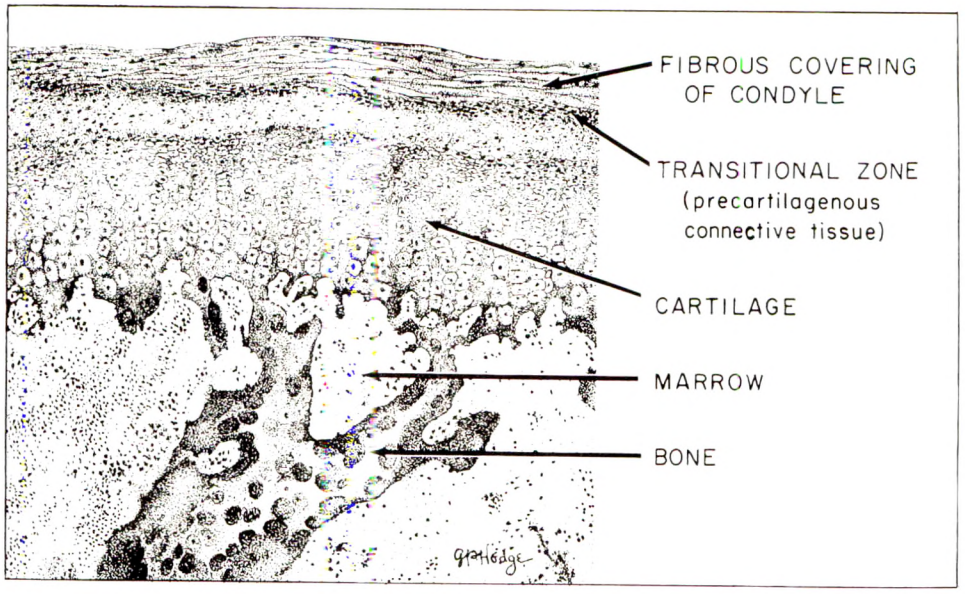


FIG. 26. Drawing of histologic section of the condylar growth center of the mandible. Growth at this site occurs by endochondral bone formation and the cartilage is derived by apposition from a layer of precartilaginous connective tissue which lies beneath the fibrous articular surface. (After Weinmann and Sicher.[53] From Orban: Oral Histology and Embryology.)

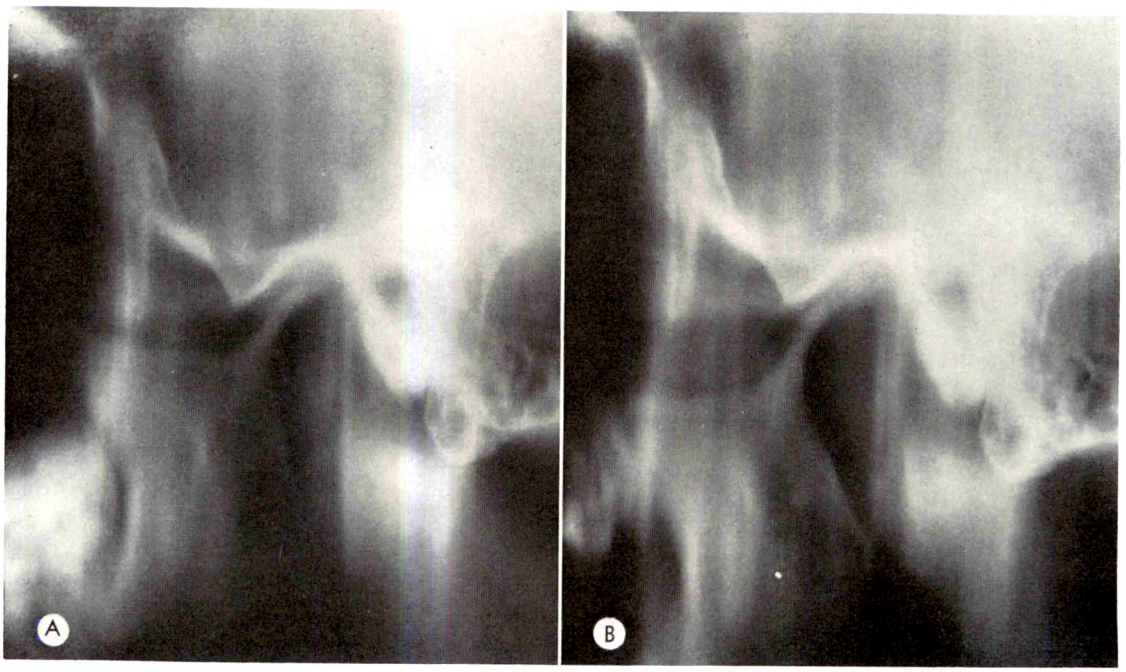


FIG. 27. Aged 22. Temporomandibular joint. (*A*) Closed mouth; (*B*) open mouth. The vertical rami are remarkably short. The condyle is tiny but its shape is more or less retained and the fossa appears normal. Note wide mandibular notch. The opposite joint had an identical appearance. No history of arthritis in these joints.

and joint destruction, that even minimal arthritis of these joints can affect the growth center, if occurring early enough in life. Temporary cessations in condylar growth are not compensated and a significant period of arrest will be reflected in the final size of the mandible.[8] One can postulate, too, that condylar growth may be affected by an adjacent inflammatory process, for micrognathia has apparently been noted as a sequel to chronic middle ear infection.[8] In this connection it is of interest that 10 of the 15 patients with micrognathia had cervical spondylitis and roentgenograms of this region were not available in 3 of the remaining 5. The possibility of a systemic or hormonal effect on mandibular growth may also be raised but there is, to our knowledge, no evidence of a hormonal deficiency in these children.

Spondylitis in juvenile rheumatoid arthritis predominantly affects the cervical spine, as in the adults with this disease.[1,4,16,18,35,36,60] Children more often than adults tend to develop apophyseal ankylosis and, in this respect, bear a resemblance to patients with ankylosing spondylitis. However, these cervical lesions differ from the latter in that paraspinal ossifications are conspicuously absent and in most cases the sacroiliac joints are normal even when traced into adulthood Occasionally, the sacroiliac joints may be affected in adults with classical rheumatoid arthritis. Consideration of the clinical as well as roentgenologic features in such cases may permit a distinction from ankylosing spondylitis.[36] However, in children with rheumatoid arthritis and sacroiliac involvement, it is often impossible to distinguish the two diseases until later in life if spinal changes characteristic of ankylosing spondylitis develop. This is particularly true because rheumatoid arthritis in children differs from that in adults in many clinical respects.[15,18,23,38,46] The difficulties in evaluating minimal sacroiliac joint findings, particularly in children, are well known. Nevertheless, sacroiliac arthritis apparently does occur in some children with rheumatoid arthritis,[4,23,33,60] as well as in those who finally develop ankylosing spondylitis.[13,19] It is our impression that the latter is quite uncommon in childhood, especially before puberty. There is little evidence in this group of patients to support the idea that spondylitis, descending to involve the sacroiliac joints, is a common sequel in juvenile rheumatoid arthritis.

The cervical spondylitis has a characteristic appearance in children. There is an outstanding predilection for the upper cervical segments and subluxations, particularly atlanto-axial, are common.[1,16,33,35,54] While spondylitis of the upper segments, with sparing of the lower, may be observed, we have not yet noted the reverse. With apophyseal ankylosis there occurs a dwarfing and alteration in form of the adjacent vertebrae and disks, presumably due to growth disturbance, "nonuse" atrophy and remodeling. The correspondence of these changes with the levels of ankylosis suggests a causal relationship. Occasionally, a similar appearance may be observed in the dorsolumbar spine suggesting that the adjacent apophyseal joints might also be affected. Grokoest *et al.*[23] described 2 patients with normal sacroiliac joints who demonstrated clinical evidence of fusion of the entire spine with diminished chest expansion. In our 4 patients (Table IV) the reduced size of the dorsal vertebral bodies was associated with reduction in the height of the adjacent intervertebral disks. This is to be distinguished from compression fractures where disk height is characteristically maintained.

SUMMARY

Certain roentgenologic manifestations of juvenile rheumatoid arthritis differ from the adult form of the disease. The distribution of involved joints, the relatively late destruction of articular cartilage and bone, and the tendency toward periosteal bone apposition and peripheral joint ankylosis are characteristic. Compression fractures of the spine and epiphyses of the extremities are frequent and hip dislocation is not uncommon. The carpal ossification centers were completely resorbed in one

case. Band-like zones of metaphyseal rarefaction may simulate those seen in childhood leukemia. Digital arterial calcification was an unexpected finding. The spondylitis is distinctive in its predilection for the cervical spine and tendency toward apophyseal ankylosis. Subluxations, particularly atlanto-axial, are common. Growth disturbances are frequent and often severe and represent a hallmark of the disease in children.

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ACQUIRED AND CONGENITAL ABSENCE OF THE ODONTOID PROCESS*

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ABSENCE of the odontoid process is considered to be a rare anomaly with only 22 cases reported in the literature. All reported cases have been classified as being congenital in origin,[6] and to the best of our knowledge, no reference has been made to an acquired loss of the odontoid process. Most of the cases described in the literature had been discovered in adults. We wish to report 4 additional cases of congenital absence of the odontoid process, all in children and seen within a period of one year, as well as one unique case of an acquired loss of the odontoid process.

ANATOMY AND EMBRYOLOGY

The odontoid process, an upward extension of the body of the second cervical vertebra, is formed from separate embryologic anlages.[2] The base is formed from 2 ossification centers which appear at approximately the sixth fetal month and fuse at the time of birth. This creates a wedge-shaped process which extends superiorly behind the anterior ring of the atlas. The apex of the odontoid process has a separate ossification center which does not visualize roentgenographically until the second year of life. Fusion of the base and the apex usually occurs by the age of 12 years. The formed odontoid process remains separated from the body of the axis by a cartilaginous disk and does not normally fuse until the adult years. A dens which fails to fuse with the body of C 2 is called an os odontoideum or third condyle.[14]

The atlanto-axial articulation is classified as a screw joint, with the central axis of motion being vertical through the odontoid and horizontal around the odontoid. The lateral articular facets of the axis are somewhat convex so that the lateral motion is not horizontal but curvilinear. Lateral rotation is checked by the alar ligaments passing from the odontoid to the occiput as well as by the atlanto-axial ligaments (Fig. 1). The curvilinear lateral motion, when combined with a vertical rotation, results in a movement like that of a screw.[5,9,13]

REPORT OF CASES

CASE I. For a period of 1 month this 27 month old white girl held her head to the left and refused to turn her neck. The child had been a breech delivery and mental retardation had been recognized at 8 months of age. On physical examination the child was found to be irritable, somewhat mentally retarded, and she absolutely refused to rotate her head, maintaining a position with her head fixed to the left. The remainder of the physical examination, including neurologic and routine laboratory studies, was normal. Roentgenograms of the cervical spine revealed an abnormal odontoid process with the presence of a displaced third condyle (Fig. 2, *A* and *B*). The child was treated conservatively and when discharged was asymptomatic.

CASE II. The parents of this 8 month old boy had noted, over a period of 48 hours, that he persisted in holding his head to the left. In addition, it was stated that the child produced unusual clicking sounds when moving his neck. A physical examination revealed an 8 month old boy who was somewhat irritable and who held his head rigidly to the left. Tight sternocleidomastoid muscles were noted on the left. The remainder of the physical examination was within normal limits. No neurologic abnormalities were encountered. Cervical spine roentgenograms revealed an absent odontoid process (Fig. 3, *A* and *B*). The infant was treated conservatively and upon discharge was asymptomatic.

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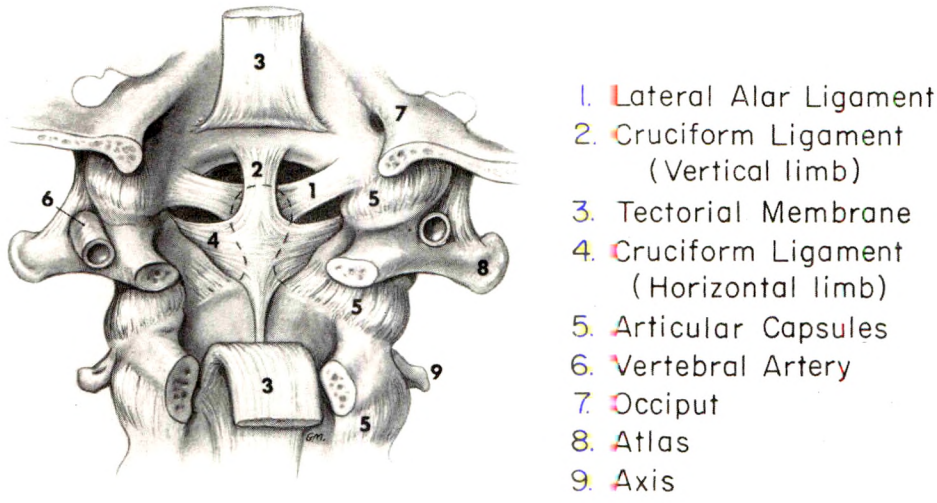


FIG. 1. Composite drawing of the upper cervical spine area with the major anatomic structures of this region labeled.

CASE III. Four days prior to admission to the hospital, this $6\frac{1}{2}$ year old girl fell, striking the back of her head. Subsequently, she complained of a stiff neck and limited motion of her head. Physical examination revealed a well-developed youngster in no acute distress. The long muscles of the neck were held in spasm and motion was limited in all directions. There were no neurologic findings and the routine laboratory studies were normal. Roentgenograms of the cervical spine (Fig. 4, *A* and *B*) showed a poorly developed odontoid process and the presence of a third condyle. The child responded to conservative management. Due to the fear and likelihood of cord compression, it was elected to do a fusion between C1, 2, and 3 of the cervical

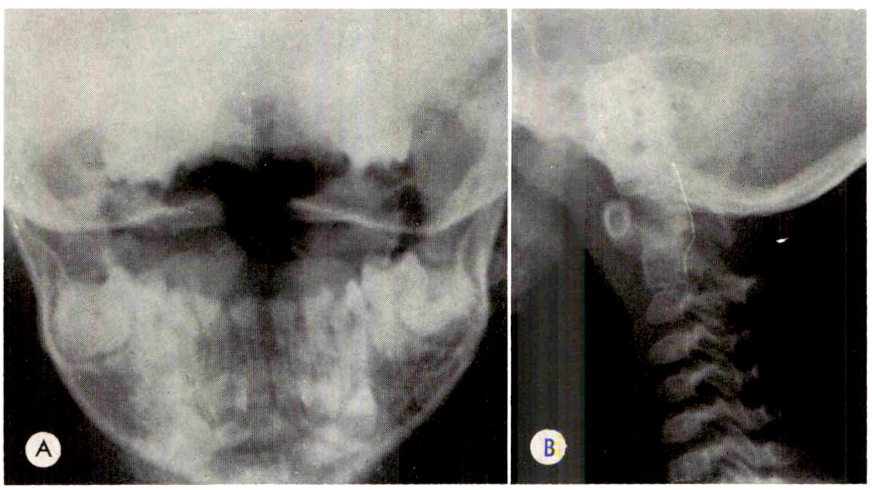


FIG. 2. Case I. (*A*) Open-mouth and (*B*) lateral roentgenograms of the cervical spine showing the abnormally developed odontoid process and the displaced third condyle.

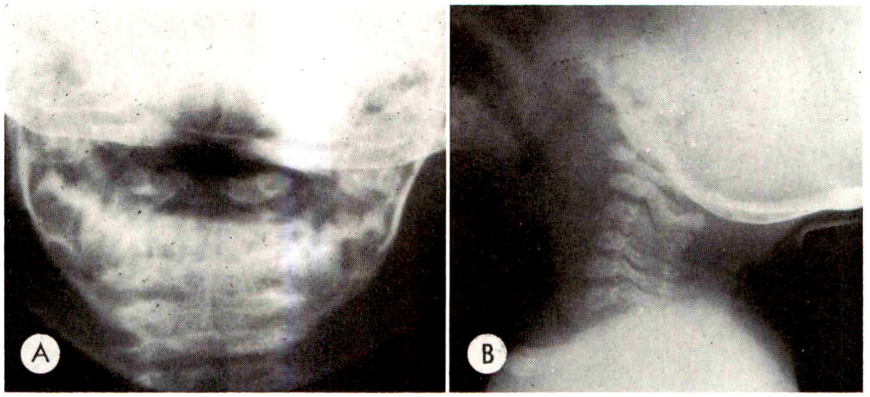


FIG. 3. Case II. (*A*) Open-mouth and (*B*) lateral roentgenograms showing the absence of the odontoid process. The poor alignment of C1 and C2 is illustrated.

spine. The procedure was accomplished without incident and no neurologic complications were encountered. The child has remained asymptomatic.

CASE IV. For the past 3 months the parents of this 10½ year old girl had noted that she turned her right foot inward. Also, when walking, she did not seem to have good control of her right arm and leg. She complained mildly of neck pain. On examination, there was muscle weakness on the entire right side. The only positive neurologic finding was a Babinski sign on the right. Roentgenograms (Fig. 5, *A* and *B*) showed a poorly developed odontoid process and the presence of a third condyle. After a trial of conservative therapy, it was elected to fuse the first 3 cervical vertebrae. No difficulties were encountered and this child has now remained free of any neurologic difficulties.

CASE V. This 4½ year old girl was referred to the Los Angeles Children's Hospital by her family physician with the diagnosis of primary pulmonary tuberculosis. For one week the child complained of a stiff neck. Roentgenograms of the neck, taken by the family physician, disclosed calcification over the first and second cervical vertebrae. The mother of the child was known to have active tuberculosis. The physical examination revealed an irritable white girl who kept her head flexed tightly to the right and refused to move her head or neck. There was marked spasm of the neck muscles. The remainder of the physical examination, including the neurologic study, was normal. The sedimen-

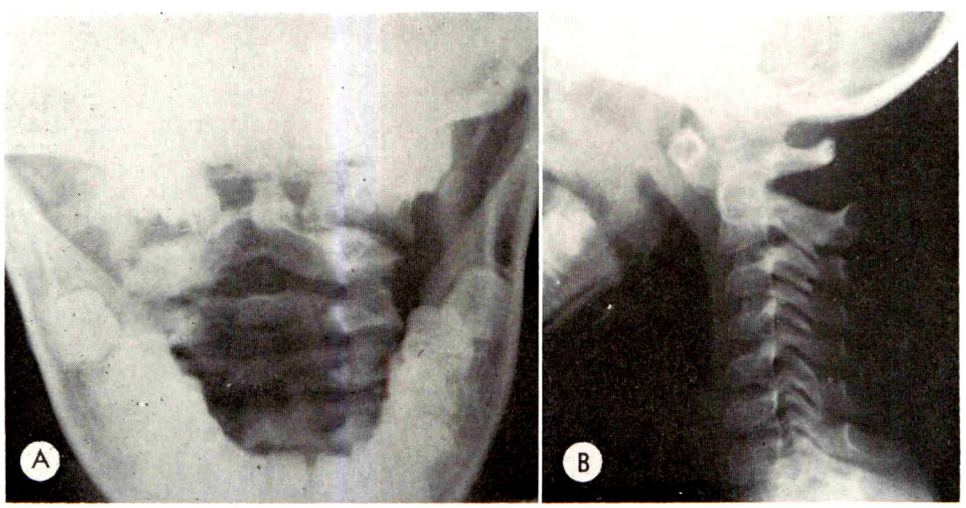


FIG. 4. Case III. (*A*) Open-mouth and (*B*) lateral roentgenograms showing the third condyle and the increase in distance between C1 and C2.

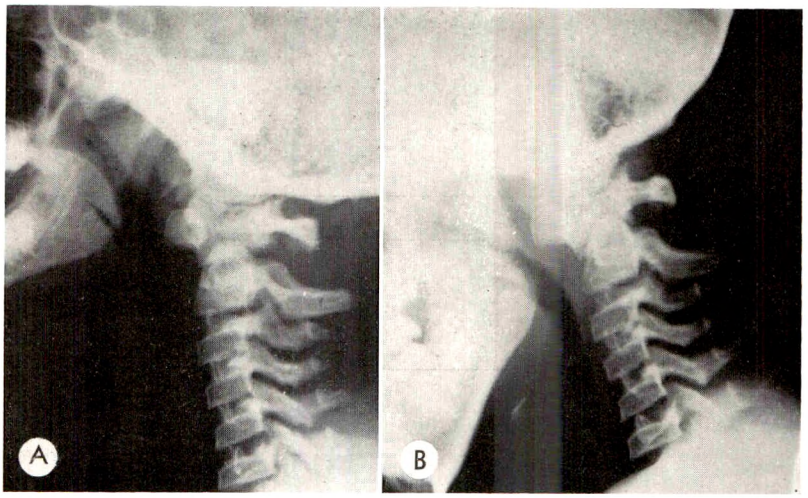


FIG. 5. Case IV. Lateral roentgenograms of the cervical spine in neutral (*A*) and mild flexion (*B*) positions show the change in relationship of C1 and C2. The third condyle is visualized.

tation rate was 31.0 mm. per hour and the old tuberculin skin test was positive. Chest roentgenograms revealed a tuberculous infiltration of the right lung. A roentgenogram (Fig. 6) of the cervical spine disclosed a destructive process of the odontoid with subluxation of the atlas to the left and anterior to the axis. The patient was treated conservatively with neck brace and chemotherapy. Subsequent sedimentation rates became normal and the patient became asymptomatic. Progress roentgenograms of the neck revealed the disappearance of the odontoid process (Fig. 7, *A* and *B*).

DISCUSSION

The stability of the atlanto-axial joint is markedly impaired in cases where the odontoid process is either absent or where there is a third condyle. The two lateral articulations are intact allowing curvilinear horizontal motion; however, the atlanto-dental joints are absent and attachments for the transverse ligament, the cruciate ligament, tectorial membrane, alar ligaments and the apical-dental ligaments are missing (Fig. 1). There is lack of control in the lateral horizontal motion and in vertical extension. Cord compression becomes a possibility and trivial neck trauma may result in serious neurologic disability.[1,7,14]

In 5 of the 22 reported cases of total absence of the odontoid process (Table 1) associated anomalies of the cervical spine were present.[3] Fourteen of the 22 cases had antecedent trauma to the neck necessitating roentgenographic examination of this region. Dislocation, subluxation or increased mobility of the atlanto-axial joint was noted in 15 cases. No neurologic findings were demonstrated in approximately 40 per cent



FIG. 6. Case V. Lateral roentgenogram of the cervical spine showing the calcific deposit in the region of C1–C2.

TABLE I

ANOMALOUS FINDINGS IN REPORTED CASES

Absence of Odontoid Process

Source	Date	Age	Sex	Trauma	Dislocation	Neurologic Complaints	Other Anomalies	Treatment
Roberts[10]	1933	20	M	Yes	Yes	None		Conservative
List (Case 5)	1941	35	M	No	No		Klippel-Feil syndrome; anomalous foramen magnum	
Weiler[18] (Case 1)	1942	14	F	Yes	Yes	None		Fusion
Weiler[18] (Case 2)	1942	12	F	No	Yes	None		Conservative
Scannel[12]	1945	23	M	Yes	Yes	Transient weakness of all extremities		Conservative
Lombard *et al.*	1945	14	F	Yes	Yes	None. Surgery. Death	Klippel-Feil syndrome	
Ivie[4]	1946	23	M	Yes	Yes	Transient weakness of all extremities		Conservative
Nievergelt[8] (Case 1)	1948	28	M	Yes	Yes	Transient left-sided weakness, positive Horner and Babinski sign		Conservative
Nievergelt[8] (Case 3)	1948	10	M	Yes	Yes	None		Conservative
Alajouanine	1949	47	M	No	Yes			
Stiefel[16]	1950	24	F	No	Yes	None		Conservative
Miyakawa[7]	1952	29	M	Yes	No	Transient brachial plexus paralysis, paresthesia		Conservative
Raskin	1953	27	F	No	No	Grand mal, epilepsy, Klippel-Feil syndrome with platybasia. Death due to herniation of brain		Conservative
Fullenlove[2]	1954	56	M	Yes	No	None		Conservative
Schultz *et al.*[15]	1956	29	M	No	Yes	Quadriparesis and pyramidal signs, thickened arch of C2, arachnoiditis—relieved by surgery		Conservative
Schiller and Nieda[14]	1957	33	M	Yes	No	Transient cervical trauma., no neurologic sequelae, rheumatoid arthritis		Conservative
Rowland *et al.*[11] (Case 1)	1958	59	M	Yes	Yes	Spastic quadriparesis, more on right, exaggerated reflexes, positive Horner and Babinski sign		Conservative
Rowland *et al.*[11] (Case 11)	1958	55	M	Yes	No	Difficulty in walking, stiffness of legs, exaggerated reflexes, positive Horner and Babinski sign		Conservative
Gillman[3]	1959	42	M	Yes	Yes	None		Conservative
Teplick *et al.*[17]	1960	42	M	No	Yes	None		Conservative
Mann *et al.*[6]	1960	59	M	Yes	Yes	Klippel-Feil syndrome, C2, C3. Death due to cord compression. Autopsy		
Present Authors (Case 11)	1961	8 mo.	M	No	No	None		Conservative

TABLE I—(*Continued*)

Source	Date	Age	Sex	Trauma	Dislocation	Neurologic Complaints	Other Anomalies	Treatment
				Os Odontoideum—Third Condyle				
Nievergelt[8] (Case 4)	1948	41	F	No	No	None, os odontoideum		Conservative
Ford[1]	1952	17	M	No	Yes	Syncope, vertigo, visual disturbance. Relieved by cast and fusion		Fusion
Flemming and Hodson	1955	55	M	Yes	Yes	None		Conservative
Sarasin	1952	24	F	No	Yes	Rt. paresis, vertigo, headache, rt. sensory motor paresis. Death		
Present Authors (Case I)	1961	$2\frac{1}{4}$	F	No	Yes	None, mental retardation		Conservative
Present Authors (Case III)	1961	$6\frac{1}{2}$	F	Yes	No	None. Treatment: fusion C1, 2, 3		Fusion
Present Authors (Case IV)	1961	$10\frac{1}{2}$	F	No	Yes	Rt. paresis. Treatment: fusion C1, 2, 3		Fusion
				Partial Absence of Odontoid Process—Hypoplasia				
Lombard (Case I)	1939	8	M	Yes	Yes	None		Fusion
Nievergelt[8] (Case 2)	1948	$7\frac{1}{2}$	F	Yes	Yes	None, rheumatoid arthritis		Conservative
Sarasin	1952	55	F	F	Yes	Radicular pain of rt. arm, associated osteoarthritis		Conservative
				Acquired Absence of Odontoid Process				
Present Authors (Case V)	1961	$4\frac{1}{2}$	F	No	Yes	None, cold abscess in C1 area with disappearance of odontoid process		Conservative

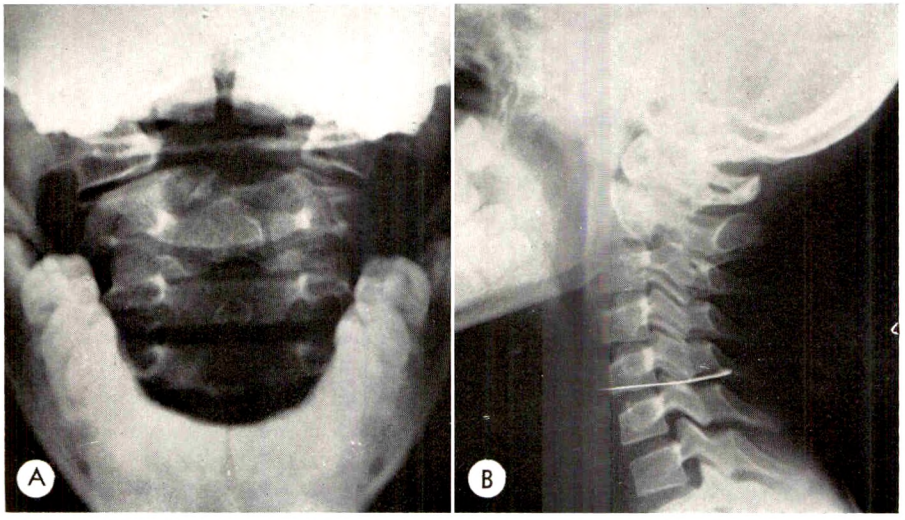


FIG. 7. Case V. (*A*) Open-mouth and (*B*) lateral roentgenograms of the cervical spine made 7 years after Figure 6 showing the absence of the odontoid as well as the malalignment of C1 in its relationship to C2.

of these cases. The remainder showed changes that varied from minor transient symptoms to death due to cord compression.

Schiller and Nieda[14] classified malformations of the odontoid process as: (1) complete absence, (2) partial absence, and (3) separation or nonfusion (os odontoideum). It was their opinion that all 3 forms led to an unstable atlanto-axial joint with potential pressure on the cervical cord or medulla. It was also thought that an absent odontoid process, as an isolated finding, was not too serious and that the possibility of permanent central nervous system damage would be unlikely. On the other hand, the presence of an os odontoideum appeared to be more hazardous. This view has been held by several authors.[3,14]

Sixteen of the reported cases with total absence of the odontoid process were treated conservatively with good results. Two were treated with fusion of the cervical spine (one of which died at surgery) and one other had excellent results after laminectomy and lysis of adhesive arachnoiditis. Our Case II with total absence of the odontoid process also was successfully managed by conservative methods.

No neurologic symptoms have been recorded in the 3 cases reported as hypoplasia of the dens (Table I). One of these had surgical fusion and 2 were managed conservatively.

Of the 7 cases in which a third condyle was present (Table I), only 3 had severe neurologic symptoms or signs. One of these experienced cerebral symptoms due to vertebral artery compression.[1] Three of the 7 had surgical fusion while 3 were treated conservatively and there was one death prior to any form of therapy. It must be recalled that the apex and base of the odontoid process fuse at approximately 7 years of age but that the base does not fuse to the body of C2 until adult life. This situation is normal and must not be construed as an os odontoideum unless the odontoid is displaced. It is conceivable that trauma to the neck, particularly during early life, could readily produce shearing of the odontoid process and cause separation to occur. Further study of this appears warranted.

Case V with acquired loss of the odontoid process is unique (Table I). It appears that this resulted from a cold abscess developing in the C1–C2 area. The positive tuberculin test and the known contact with tuberculosis makes this diagnosis seem a very strong likelihood. The infectious process occurred during the developmental stage of the odontoid and this could play a role in the selective loss of this structure.

SUMMARY

Five additional cases of absence of the odontoid process or of the presence of a third condyle have been reported. All of these occurred in children and 4 were seen in a period of less than one year. The literature has been reviewed and reported in tabular form.

The first reported case of an acquired absence of the dens is included, the apparent etiology being an infectious process, probably tuberculosis, involving the C1–C2 region.

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HYPOPARATHYROIDISM: A REVIEW OF THE LITERATURE AND REPORT OF TWO CASES IN SISTERS, ONE WITH STEATORRHEA AND INTESTINAL PSEUDO-OBSTRUCTION*

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BEUMER and Falkenheim[5] first reported a case of idiopathic hypoparathyroidism in 1926. In 1939, Drake *et al.*[12] established their criteria for the diagnosis of this condition. These criteria were that: (1) the serum calcium level should be low; (2) the level of serum inorganic phosphorus must be high; (3) renal insufficiency, steatorrhea, chronic diarrhea, and alkalosis must not be present; and (4) osteomalacia and rickets should be excluded by roentgen examination.

Steinberg and Waldron[35] in 1952, using the criteria of Drake *et al.*,[12] found 51 cases in the literature and added one of their own. In addition to the criteria of Drake *et al.*,[12] they also required that the serum inorganic phosphorus be greater than 7 mg. per cent when the age was below 16 years. Bronsky *et al.*,[6] in another review article, emphasized the necessity of a phosphate diuresis following the administration of a potent parathyroid extract or a therapeutic response to adequate intramuscular administration of this hormone as a requirement for making a diagnosis of idiopathic hypoparathyroidism.

It is the purpose of this communication to report 2 cases of hypoparathyroidism and to review certain roentgenographic aspects of the disease.

ROENTGENOGRAPHIC FINDINGS

A. *Skull and Brain.* Thickening of the skull tables and homogeneous thickening of the petrous bone and even thickening of the facial bones to a lesser degree may be present.[1,15,37,40] Evidence of increased intracranial pressure such as widening of sutures may be found.[21,36]

The pathologic demonstration of calcification in the brain was seen as early as 1902, but roentgenographic demonstration was first reported in 1939.[13] In the earlier stages, the calcifications are discrete, but later coalesce into a rather homogeneous mass.[13,33] The calcification in the region of the basal ganglia may be in the form of striae, wide spread, linear or punctate.[6,7,14,16,21,22,23,24,30,31,33] The calcification may rarely occur in the choroid plexus and cerebellum.[6] No improvement in the degree of calcification is expected with treatment.[13,53] Slight dilatation of ventricles has been reported.[24,40]

B. *Teeth.* Hypoplasia of enamel and dentine,[2,32,34] blunting of the roots,[38] delayed or failure of eruption,[6,14] thickening of the lamina dura, and prominence of the dental membrane[15,18] are some of the dental abnormalities in this disease.

C. *Digestive System.* Gastrointestinal spasm, hypersecretion, and spasm of the antrum are the main manifestations.[3]

D. *Bones.* In 44 cases reviewed by Bronsky *et al.*[6] 9 per cent had generalized and 23 per cent had localized increased bone density. Bone density was decreased in 10 per cent of the cases. In 57 per cent of the cases the structure and/or the density were normal. In Steinberg and Waldron's[35] series comprising 52 cases, of 22 patients in whom skeletal roentgenograms were mentioned, 17 were reported as normal, 4 showed a generalized increase in bone den-

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sity and one case revealed diminished bone density.

Other bone manifestations reported in conjunction with idiopathic hypoparathyroidism are: (1) deformed hip joint with thickening and sclerosis of the femoral head and acetabulum plus narrowing of the joint space;[1] (2) subcutaneous calcification;[35] (3) premature closure of the epiphyses;[6] and (4) calcification of the ligaments of the spine.[31]

REPORT OF CASES

Case 1. S.K. (Hospital #28-94-15) is a 16 year old white female. She was admitted to Children's Memorial Hospital with a history of tetany and convulsions since the age of 3 years. Occasional bouts of vomiting and the presence of bits of undigested foods in her stool since she was 7 years of age were interesting features in her past history. She was admitted to another hospital in July, 1959, at 14 years of age with the chief complaint of recurrent vomiting and loss of weight. She had abdominal distention and absent bowel sounds. The serum calcium was 4 mg. per cent and the serum phosphorus was 5.2 mg. per cent. She responded favorably to treatment with dihydrotachysterol, calcium gluconate, etc., and was discharged on August 26, 1959, much improved. Two more admissions to that hospital became necessary due to the continuation of vomiting. At the third admission, because of persistence of vomiting, an abdominal exploration was undertaken and no definite obstruction was found. Duodenojejunostomy and appendectomy were performed; however, postoperatively, the vomiting continued. At re-exploration no definite obstruction was recognized. The vomiting and abdominal distention persisted and she was transferred to Children's Memorial Hospital on October 8, 1959.*

Physical Examination. At admission, the patient was markedly dehydrated and malnourished. Her weight was 23.2 kg. (51 pounds) and her height was 137 cm. (54 inches). She had lost 17.7 kg. (39 pounds) since January, 1959. Vomiting was the main problem.

Marked abdominal distention and absent bowel sounds were the outstanding findings. Carpopedal spasm and positive Chvostek sign were present. Her hair was dry, coarse, and sparse. Her teeth were in poor condition with multiple caries. The joints of the elbows, wrists, fingers, knees and ankles were prominent and articular motions were limited. This was most marked in her hands resulting in an inability to make a fist. A slit lamp examination revealed bilateral subcapsular lens opacities.

Laboratory Findings. The hemoglobin was 9.0 gm. The serum calcium was 6.4 mg. per cent and repeated determinations ranged from 6.2 to 8.4 mg. per cent (normal 9.0 to 11.0 mg. per cent). The serum phosphorus was 4.6 mg. per cent, repeated determinations ranged from 6.9 to 8.2 mg. per cent (normal 4 to 6.5 mg. per cent). The serum alkaline phosphatase was 5.3 Bessie-Lowry units (normal 2.8 to 6.7). The blood urea nitrogen ranged from 11 to 17 mg. per cent. The Ellsworth-Howard test with 200 units of parathyroid hormone intravenously showed an almost doubled phosphorus excretion in the urine (from 34 to 84 mg. per hour). The response in the control was four-fold (from 20 to 80 mg. per hour). Adrenal function studies were reported as normal. The duodenal enzyme activities were considered within the normal range. Fat in her stools (Sudan IV stain) ranged from 1 plus to 3 plus (2 plus or above is considered abnormal). The radioactive fat absorption was 0.51 per cent at 6 hours (normal 12–15 per cent).

Roentgenographic Findings. The bones were generally osteoporotic. No evidence of osteomalacia was detected. No abnormal intracranial calcifications were seen. In the upper extremities (Fig. 1) a small flake of calcium was seen in the right shoulder region. Undertubulation of the proximal humeri was present. The proximal epiphyses of the radii were abnormally formed and were somewhat flat. The hands were short and relatively broad. The long bones of the hands were short and undertubulated. These features were most prominent in the metacarpals. A spina bifida occulta of the seventh cervical vertebra was present. The proximal epiphyses of the femora were flat and the femoral necks were wide. The abnormal tubulation of the other long bones in the lower extremities was of a lesser degree than that in the upper extremities (Fig. 2).

There was a lack of sharpness of the lamina dura which was in some areas almost invisible roentgenographically. The teeth were widely spaced and were of poor quality. The dentine was rather dense and chalky in appearance.

* We are indebted to Dr. Joe Spann, Tulsa, Oklahoma, for referring this patient.

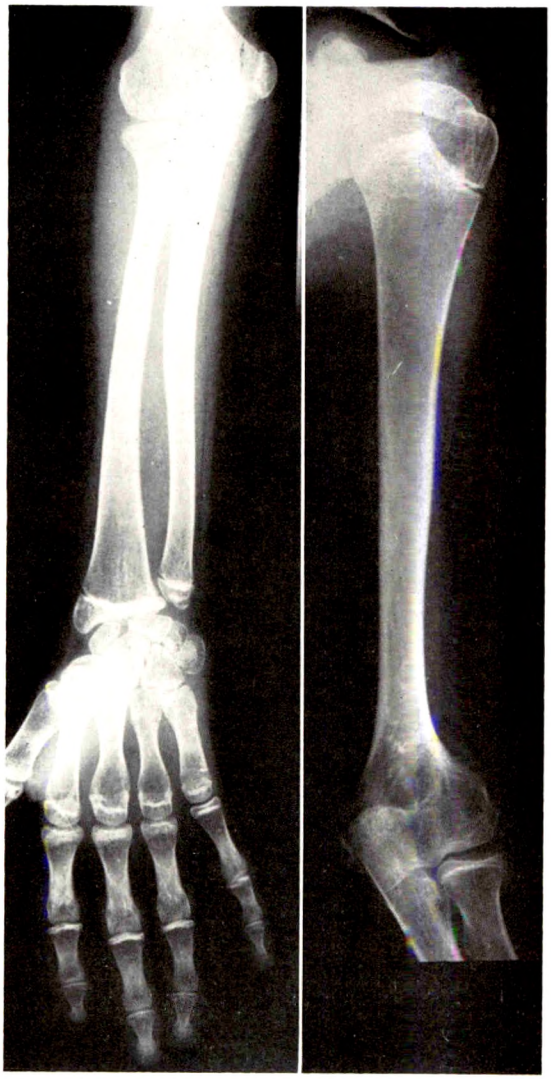


FIG. 1. Case 1. Osteoporosis of the long bones, a small flake of calcium in the shoulder region, the relatively flat proximal epiphysis of the radius and the short and broad hand are shown. Note particularly the undertubulation of the metacarpals.

There was some narrowing of the pulp space. The third molars, at 15 years of age, were not yet mineralized (Fig. 3).

Several roentgenograms of the abdomen were taken after her admission to the Children's Memorial Hospital. Moderate to marked degree of distention of what was considered to be small and large intestines was found repeatedly (Fig. 4). A barium enema examination on November 30, 1959 (53rd hospital day) showed no definite abnormalities and, in particular, no dilatation was found. A small bowel study on January 12, 1960 (96th hospitalization day) (Fig. 5*A*) demonstrated the abnormal mucosal pattern of the proximal jejunum with fragmentation of the barium column and absence of the fine mucosal folds. The follow-up roentgenograms taken at 8 hours after the intragastric injection of the contrast medium (Fig. 5*B*) showed dilution of the barium suspension in the dilated loops of small bowel with a very slow progression of the barium meal. The roentgenograms taken at 20 hours (Fig. 5*C*) demonstrated similar findings; barium had not reached the large intestine. On the

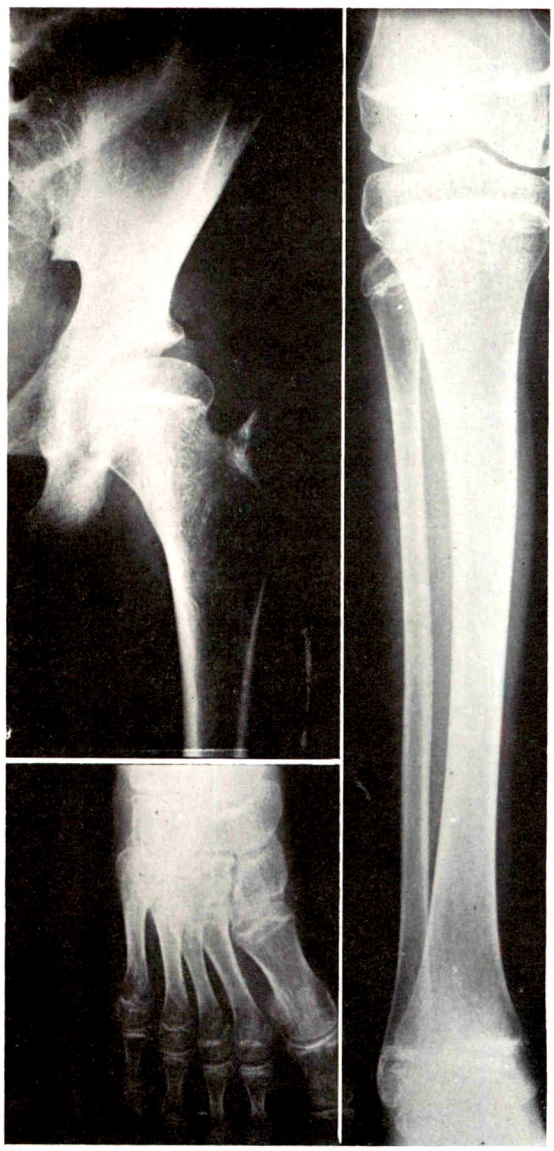


FIG. 2. Case 1. Osteoporosis, flatness of the femoral head and the wide femoral neck are demonstrated.



Fig. 3. Case 1. Mandibular roentgenogram showing a lack of sharpness of the lamina dura of the first molar, the widely spaced teeth, the oblique angle of an unerupted second molar, the lack of calcification of the third molar and the narrowness of the pulp space.

roentgenograms taken at 48 hours (Fig. 5*D*), some barium was seen in the colon but the major portion remained in the small bowel. Air fluid levels were seen in the small bowel. The opacified portion of the large bowel was not distended and the haustral markings in the ascending colon were not remarkable. On the basis of this examination, in particular the lack of dilatation of the large bowel, the possibility of a partial intestinal obstruction in the distal small bowel was suggested and operative procedures were undertaken (see hospital course). A repeat small bowel study of February 10, 1961, 13 months later, failed to show distention of the small and large bowels. The mucosal pattern of the small intestine remained coarse, but much improved since the examination of January, 1960 (Fig. 6, *A* and *B*). In the roentgenogram taken at 4 hours after the ingestion of barium meal, most of the contrast medium was noted to be in the colon.

Hospital Course. Following admission to Children's Memorial Hospital, the patient was treated for dehydration, salt deficiency (including potassium) and hypoparathyroidism with combined therapy; however, vomiting and abdominal distention persisted. On the 34th hospital day adrenocortical therapy was started and on the 54th hospital day ileus was no longer present and oral feeding became possible. Due to the development of edema the dosage of corticosteroid was gradually reduced. On the 76th day of her hospitalization, she developed septicemia, vomiting, and ileus. The corticosteroid therapy was discontinued and the ileus persisted.

On the 102nd day of hospitalization, due to continuous vomiting, distention and roentgenographic findings which were interpreted as partial intestinal obstruction, an exploratory laparotomy was performed by Dr. William R. Richardson. This operation revealed a loop of small bowel adherent to the anterior abdominal wall at the site of the old incision. An omental band from the right side of the abdomen was adherent to a small bowel loop in the left gutter. The terminal ileum was noted to be thick walled and smaller in caliber than the rest of the small intestine. A milky "congealed" glistening coat-

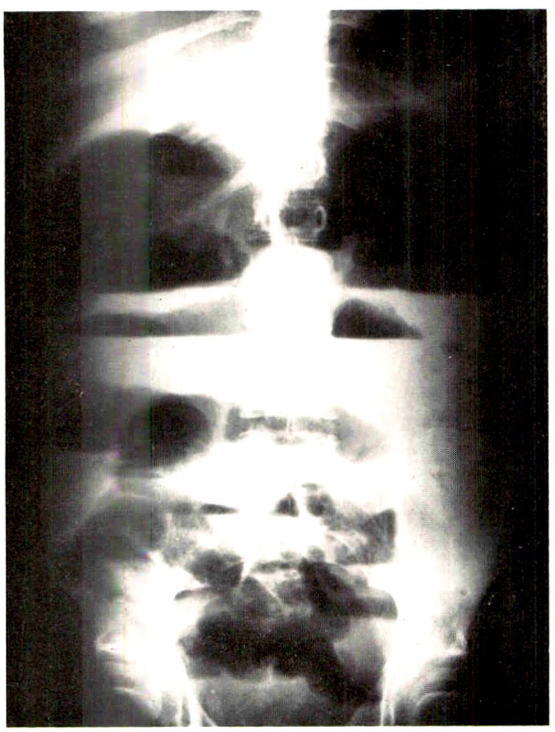


Fig. 4. Case 1. Intestinal distention and multiple air fluid levels are demonstrated.

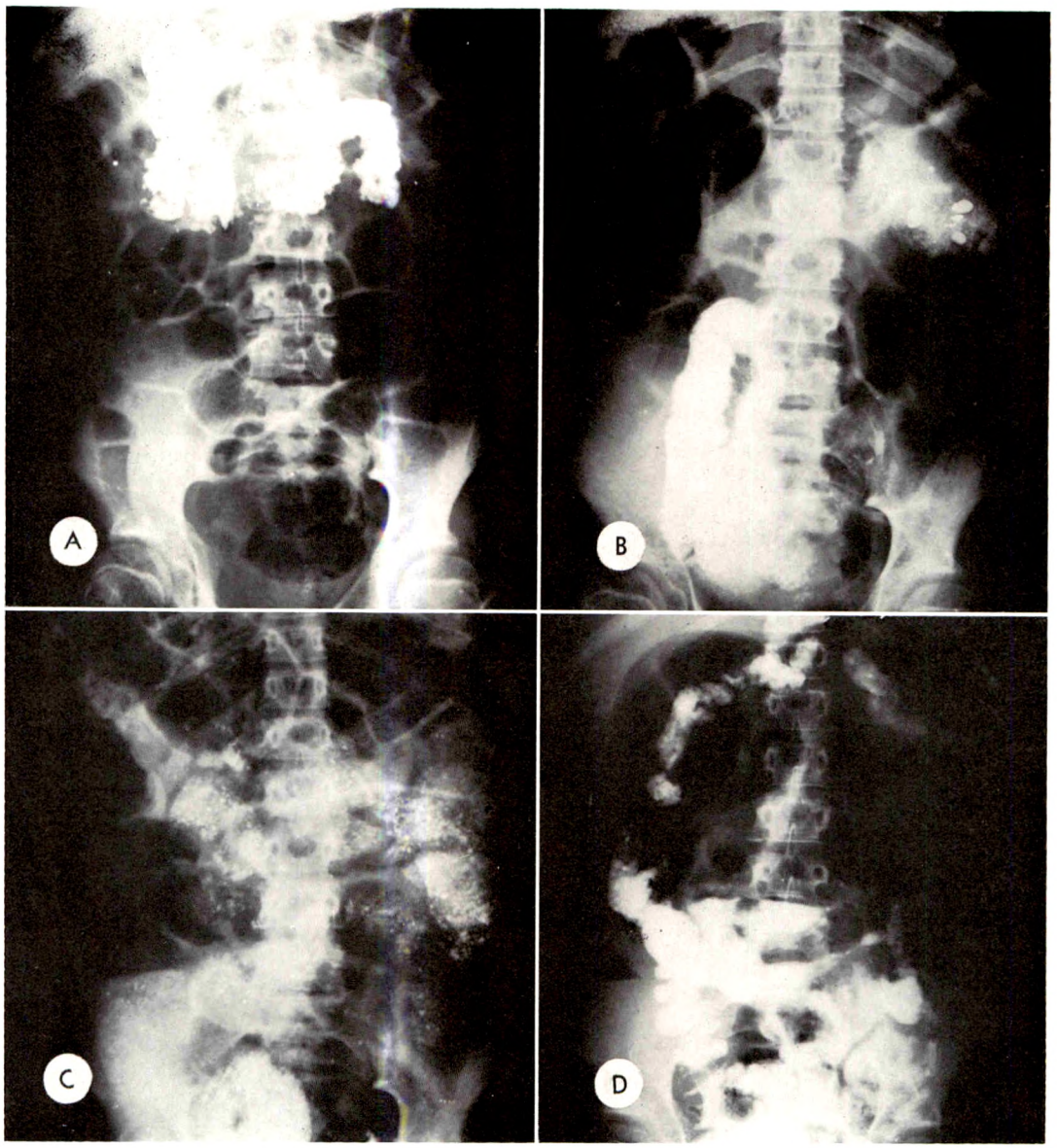


FIG. 5. Case 1. Roentgenograms showing (*A*) the abnormal mucosal pattern of the proximal jejunum, (*B*) fragmentation of the barium column and dilution of barium suspension in the distal small intestine at 8 hours, (*C*) the lack of barium in the large intestine at 20 hours, and (*D*) opacification of the nondistended colon at 48 hours with a significant amount of barium still remaining in the small bowel.

ing was present. No clear cut point of mechanical obstruction was found (Fig. 7). The adhesions were lysed from the anterior abdominal wall and also from the omentum. However, she continued to be symptomatic with distention and required gastric suction to prevent vomiting. On the 121st day of hospitalization, two surgical procedures were combined. The first was a rectal biopsy to confirm the presence of ganglion cells, and the second was a laparotomy. The distal ileum seemed to be in marked spasm. In addition, the small bowel and the mesentery were thickened, erythematous and edematous. The mesentery of the small bowel was filled with numerous 1 cm. sized lymph nodes. The small bowel and the mesentery had the appearance of being covered with a whitish milk-like exudate. Resection of a short segment of the distal ileum and ileocecal junction was performed, with construction of a double-barrel ileocolostomy.

The pathologic examination was done by Dr.

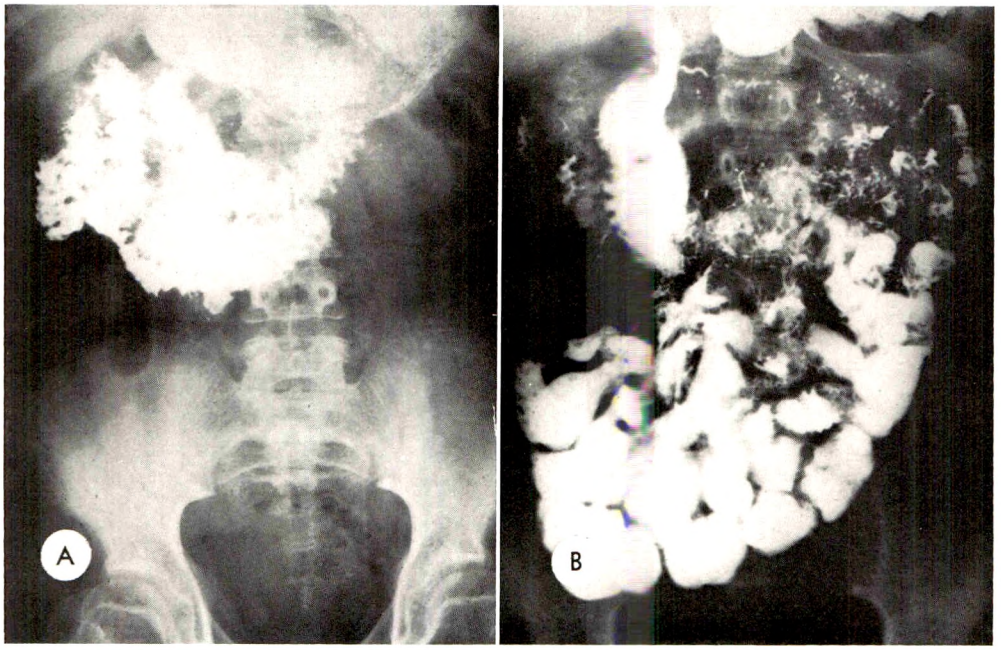


FIG. 6. Case 1. (*A*) Note the absence of the distention of the bowel and the persistent but improved abnormal mucosal pattern of the jejunum. (*B*) At 1 hour and 45 minutes the improvement in the small intestinal pattern is seen.

James P. Dewar. The specimen consisted of 32 cm. of ileum and 3 cm. of cecum. The microscopic report revealed that the section from the ileocecal valve showed an intact mucosa. The underlying submucosa contained innumerable lymphocytes with some follicle formation. The lymphocytic band was much deeper than usual. The underlying muscle wall showed an infiltration by a scattering of lymphocytes. Between the muscle layers numerous nerves and ganglion cells were noted. The serosal tissues were markedly congested. A section from the proximal end of the ileum showed only a scattering of lymphocytes in the submucosa and an occasional lymphoid follicle. The underlying muscle was not remarkable; the serosal vessels were congested; and lymphoid hyperplasia of the ileocecal valve and terminal ileum was present. The biopsy of the rectum was reported as essentially normal.

During the postoperative days, ileus and

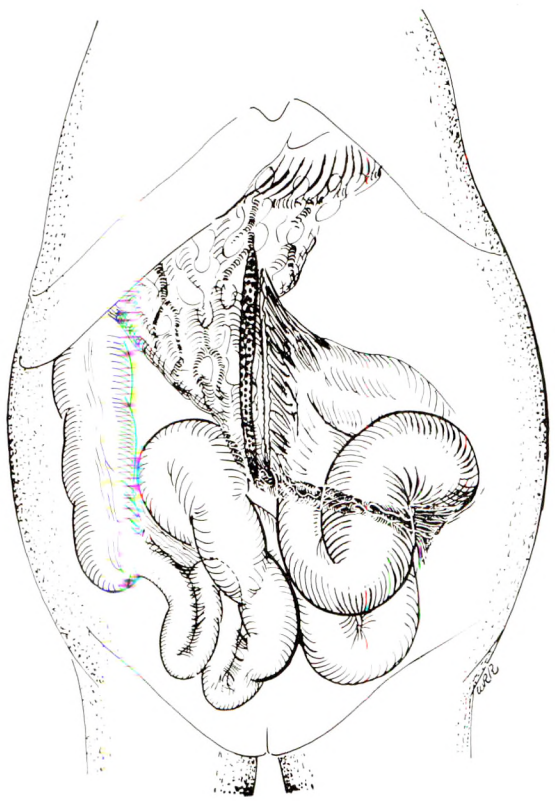


FIG. 7. Drawing showing the omental band from the right side of the abdomen extending to the left and adherent to a small bowel loop in the left gutter that was seen in Case 1. Note the narrow segment of distal ileum and the absence of an extrinsic obstructing lesion.



FIG. 8. Note the shortness of stature, the stocky body habitus, the round face, and the small and broad hands of Case I (right) at the age of 16 years and compare these features with Case II at 11 years of age (left). Two years earlier they were the same height.

vomiting persisted and corticosteroid therapy was re-instituted. She also received dihydrotachysterol and calcium gluconate orally, and a gluten free diet. There was a gradual decrease in abdominal distention over a period of one month. She was discharged on the 200th day of her hospitalization and treatment was continued at home. In July, 1960 the ileostomy was closed extraperitoneally.

Presently, almost 2 years after her original admission, her weight is 37.2 kg. (82 pounds) and her height is still 137 cm. (54 inches). Her intelligence quotient is 79. She is short, stocky, and has a round pleasant face (Fig. 8). She is sexually retarded. The tetany is well controlled. A sweat chloride study on July 1, 1961 was reported as being within the normal range.

The present treatment consists of a gluten-free diet, vitamin D_2, calcium chloride and corticosteroid.

CASE II. J.K. (Hospital #29-12-93) is an 11 year old white female who was admitted to Children's Memorial Hospital on December 1, 1959, with a history of cramps, and drawing of hands and feet of almost one year's duration. She had three convulsive seizures during the 7 months prior to this admission. No history suggesting steatorrhea was elicited.

Physical Examination. At admission, she was found to be a well nourished and well developed patient. Her weight was 37.7 kg. (83 pounds) and her height 137 cm. (54 inches). Her intelligence was considered to be normal. Multiple decayed teeth, several with brownish color spots and resorption of the crown, were noted. The Chvostek sign was positive.

Laboratory Findings. The serum calcium levels ranged from 6.0 to 7.0 mg. per cent and the serum phosphorus ranged from 6.4 to 12.3 mg. per cent. The blood urea nitrogen was 16 mg. per cent. No excess fat was detected in stools. The Ellsworth-Howard test with 200 units of parathyroid hormone intravenously showed an increase of urinary phosphorus excretion from 2 to 175 mg. per hour and was considered to be diagnostic for idiopathic hypoparathyroidism. Adrenal function tests with ACTH and sweat chloride determination were considered to be normal.

Roentgenographic Findings. No intracranial calcification was recognized. The striking finding was the marked increase in density of the metaphyseal-diaphyseal regions of the proximal humeri, distal radii, distal ulnae, distal femora and proximal tibiae. This increase in bone density had a chalky appearance with a lack of normal trabecular pattern. The diaphyseal borders of the dense segment were rather irregular (Fig. 9 and 10). The size and configuration of the hands and feet were not unusual. The dental examination at the age of 12 years revealed the presence of normal lamina dura. None of the deciduous teeth were present. A small bowel study showed no abnormalities. The transit time of the small intestine was about $1\frac{1}{2}$ hours.

Following discharge, she has been under treatment for idiopathic hypoparathyroidism with good response and there has been no recurrence of convulsions.

DISCUSSION

The clinical and laboratory findings of the second case are typical for the diagnosis of idiopathic hypoparathyroidism. On the other hand, the shortness of stature, the

stocky body habitus, the round face, the short and stubby hands of the first patient combined with only a two-fold increase in phosphorus excretion in the urine[6,26] would be more in favor of the diagnosis of pseudohypoparathyroidism. But during the course of treatment with parathyroid hormone a drop of the serum phosphorus from 6.5 mg. per cent to 4.5 mg. per cent occurred. This would be more in keeping with a diagnosis of idiopathic hypoparathyroidism. Because of this response, the definite diagnosis of idiopathic hypoparathyroidism in her sister and the statement by Bronsky *et al.*[6] that 10 per cent of the patients with idiopathic hypoparathyroidism may have abnormal body build, this diagnosis is favored.

The bone density in idiopathic hypoparathyroidism and pseudohypoparathyroidism is normal in most of the cases. Osteoporosis, which was present in the first patient, is an unusual finding in idiopathic hypoparathyroidism and pseudohypoparathyroidism but has been reported.[6,26,35] Osteoporosis has been considered to be due to a decreased intestinal absorption of calcium overshadowing a decrease in osteoclastic activity of the bones.[6] The duration of the steatorrhea in our patient is not known, but was probably of long standing and may have been responsible for the osteoporosis. The abnormal findings in the metacarpals except for osteoporosis are similar to those described by Achenbach and Böhm.[1] The shortness of the stature was obviously not due to the premature closure of the epiphyses. The very small flake of calcium in the shoulder region is similar to the findings of Steinberg and Waldron's[35] patient who had idiopathic hypoparathyroidism. Thus the presence of this localized finding alone would not be of help in the roentgenographic differential diagnosis of idiopathic hypoparathyroidism and pseudohypoparathyroidism.

The increase in bone density in the

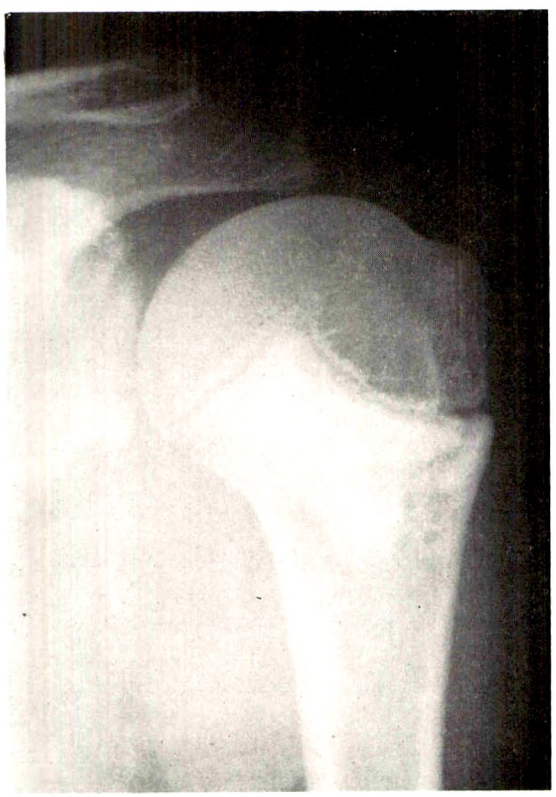


Fig. 9. Case II. The wide irregular chalky density in the metaphyseal-diaphyseal region of the humerus is seen.



Fig. 10. Case II. The metaphyseal-diaphyseal region of the distal femur and proximal tibia shows the irregular increased bone density.

metaphyseal region in Case II is a rare but expected finding. Two cases in the reviewed literature which show somewhat similar findings are reported by Achenbach and Böhm[1] and Lowe *et al.*[25] The first[1] dealt with a case of hypoparathyroidism in an adult. The most striking finding in this patient was sclerosis in the iliac crest and a double line of sclerotic bone about the borders of the vertebral bodies. Changes similar to those were seen at the shoulders and the knees in the metaphyseal regions. The widening of the dense cortex of the long bones resulted in narrowing of the marrow space. The patient of Lowe *et al.*,[25] with the diagnosis of pseudohypoparathyroidism and steatorrhea, is of special interest. Unusually dense metaphyses with an abnormal trabecular pattern characterized by mingling of dense and rarefied areas were present. A comparison with our roentgenograms shows that the width of the metaphyseal density is less than that in our second case and does not extend in the direction of the diaphysis to the same marked degree. The lack of roentgenographic visualization of the trabeculae in our case, in particular in the proximal humeri, indicates a significant degree of abnormality of enchondral bone formation.

The "Syndrome" of Steatorrhea and Hypoparathyroidism. Another interesting aspect of the first patient's clinical picture is steatorrhea. The combination of hypoparathyroidism and steatorrhea has been considered a "syndrome" by Jackson and his co-workers[19,20] and has been reported by others.[8,9,10,11,23,25,26,27,28,31,39]

Dense metaphyses and trabeculae,[25] osteoporosis,[8,9,19,39] retarded epiphyseal fusion,[27] abnormal gastrointestinal studies of steatorrhea,[9,11,19,23,39] gallbladder calculi,[23,25] and basal ganglia calcification[25,28] are the abnormal roentgenographic findings reported in patients with this "syndrome."

Pseudo-Obstruction of Small Intestine and Surgical Findings. Case I with steatorrhea and hypoparathyroidism presented the problem of pseudo-obstruction and eventually underwent 4 laparotomies. The authors are not aware of previous reports in the literature of the combination of steatorrhea, hypoparathyroidism, and pseudo-obstruction with similar operative findings. The narrowed distal ileum, without evidence of mechanical obstruction, and the small bowel distention were the two striking findings. It is possible that the narrowed distal ileum could very well be a tetanic manifestation. In support of this statement is Higgins' report.[17] One of his patients, a lactating woman with tetany, entered the hospital with a diagnosis of intestinal obstruction and a history of having twitching of the face. A gastrointestinal examination during one of these attacks showed delayed progression of the contrast medium, which took 12 hours to reach the cecum. A second examination during the quiescent period showed none of the striking features of the previous examination and there was no delay in the progression of the barium meal. However, because of the question of the presence of an associated surgical lesion, an emergency laparotomy was performed. A very marked peristaltic contraction reducing the intestine to the size of a pencil for a length of about 2 to 3 feet in the mid-portion of the small intestine was found. No resection of this segment was done. With the use of calcium, the condition of this patient improved. Naish *et al.*[29] reported 3 patients with steatorrhea, without reported evidence of hypoparathyroidism, who underwent operations for what was thought to be a mechanical obstruction. No mechanical obstructions were found at laparotomy. Marked segmented movement of the gut was visible roentgenographically and at laparotomy.

We feel that some mechanism associated with steatorrhea probably played the major role in the production of the pseudo-obstruction picture in our case for the following reasons: (1) the patient was under treatment with intravenous calcium and had no clinical evidence of tetany, and (2) there was a very satisfactory response to glucocorticoid treatment for steatorrhea.

SUMMARY

The clinical and roentgenographic findings of idiopathic hypoparathyroidism and pseudo-hypoparathyroidism are reviewed. Two sisters with idiopathic hypoparathyroidism, one with and the other without evidence of steatorrhea, are reported. The patient with steatorrhea manifested the unusual complications of severe ileus and pseudo-obstruction which resulted in several laparotomies and complications of treatment. Only when the diagnosis of steatorrhea was suspected and established and the treatment with glucocorticoids instituted did the ileus and pseudo-obstruction remit. The similarity of this unusual case to other reported cases is discussed. The second case showed a rare bony manifestation of idiopathic hypoparathyroidism.

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METAPHYSEAL DYSOSTOSIS OF MIXED TYPE IN A FEMALE CHILD

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IN 1934 Jansen[6] described a rare condition of disturbed endochondral bone formation under the name of metaphyseal dysostosis. Since then several similar cases with varying degrees of severity of the lesions in which disturbance of endochondral bone growth was limited to the metaphyses have been described.[1,3–5,7–12] Finally, Weil[12] distinguished two clinical types: the Schmidt type and the Jansen type.

The Schmidt variety of metaphyseal dysostosis is the much more common one. These patients are short of stature. The shortening concerns principally the long bones. The bones of the head are normal. The metacarpal bones and the phalanges may be somewhat shortened.[7] There are some reports about the heredity in the Schmidt variety,[7,9,10,11] which allows us to presume the presence of a dominant gene. The clinical and the differential diagnosis of this type has been described in previous papers and will not be discussed in this report.

The purpose here is to describe metaphyseal dysostosis in a female, 6 years of age. It is impossible to classify the case in one of the two existing clinical groups because there are characteristics of both the Jansen and the Schmidt varieties, but with greater similarity to the Jansen type. In the literature, we have found only 4 examples characteristic of the Jansen type of metaphyseal dysostosis. Of these, one patient reported by Gram and others,[4] a female 5 years of age, showed marked chemical changes in the blood and it is difficult to exclude the possibility that her roentgenologic findings were secondary to a basic metabolic defect, which was reflected in the abnormalities in the serum levels of calcium, phosphorus, and alkaline phosphatase activity; and also of some disturbances of renal function. In the 3 other cases only the calcium level was increased questionably above the normal value.[1,4,6] No other biochemical defects were found. For this reason it may be doubted that we have in the case of Gram *et al.* the same entity which was described by Jansen,[6] Weil,[12] and by Cameron and others.[1]

Nor can the cases described by Hühne and Schönfeld,[5] and Catel[2] be accepted as the Jansen type of metaphyseal dysostosis because there are no roentgenologic changes in the metacarpals in the first case. In the Catel case, the hands, feet, and vertebral column are normal. In both cases the serum phosphatase activity was not measured, thus hypophosphatasia and renal rickets were not excluded.

In all the patients with metaphyseal dysostosis of the Jansen type, the metaphyses of all of the tubular bones in the extremities were affected. However, in the skull and spine, ribs and clavicles ossification appeared to be normal. Of the round bones, only the calcaneal bones were dysplastic. The metaphyseal lesions were much more severe than in the Schmidt type. All the cases were sporadic and we may presume that a dominant mutant gene was responsible for the disease in all the cases since there was no consanguinity in any of the patients.

REPORT OF A CASE

A.K., a female 6 years of age, was examined at the Endocrine Outpatient Clinic of the Medical Academy in Gdansk, Poland, because of short stature.

She was the result of the first pregnancy of

* Radiologist of the First Pediatric Clinic, Poznan, Poland.
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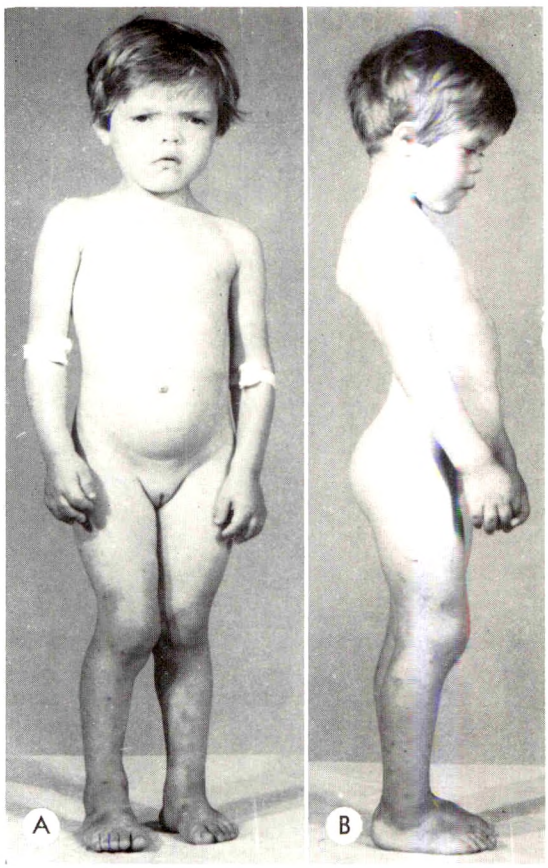


FIG. 1. Photograph of A.K. at 6 years of age. (*A*) Frontal and (*B*) lateral views. Her height is 20 per cent less than the mean value for Polish girls of 6 years. The interocular distance is conspicuously widened. The partially flexed hands are broad and stubby. Both knees deviate mediad with a corresponding separation at the ankles and feet; this knock-knee deformity is more pronounced on the right side. In the lateral view a shallow Harrison's groove is visible just below the nipple of the right breast and the spine presents a long shallow lordosis.

healthy young parents. During the entire pregnancy the mother vomited frequently and during the sixth month had pyuria. The delivery was uncomplicated. This apparently normal newborn infant weighed 2,900 gm., and her length was 49 cm.

Growth and motor development were normal during the first 2 years. She sat at the age of 6 months and walked at 14 months. The first teeth were noted at 6 months. In her third year she had measles with subsequent pneumonia and anemia. At the end of the third year she had developed knock-knees. Also, she was shorter than other children of her age, and this difference is said to have increased during the following years. She had neither pain nor walking difficulties and was normally active. Once, about 3 months before the visit to us, she did complain about leg pains which lasted for about 3 days. She was an alert child with normal mental development.

Her parents and other relatives were of normal stature.

At the time of examination at the Endocrine Outpatient Clinic the child was well nourished but short in stature; height 92 cm., weight 13.5 kg. (Fig. 1, *A* and *B*). Her head seemed to be large in relation to her stature. The arms and legs, and hands and feet were shortened. Her measurements are listed in Table I. A lumbar lordosis was present, but her gait was normal despite marked knock-knees. The distance between the medial surfaces of the ankles while standing was 8.5 cm. (Fig. 1*A*). External rotation at the hip joints was limited, but movements in all the other joints were normal. Muscular tone, volume, and strength were all normal. In the organs of the chest and the abdomen, no abnormalities were found. Harrison's groove was present (Fig. 1*B*).

TABLE I

A COMPARISON OF OUR CASE WITH A NORMAL 6 YEAR OLD FEMALE

Measurements in cm.	Our Patient	Normal
Height	92.0	114.8
Circumference of head	48.5	49.7
Circumference of chest	50.5	54.2
Circumference of abdomen	46.0	53.2
Circumference of hips	52.5	58.1
Span	90.0	113.1
Sitting height	53.0	59.7
Upper limb (acromion to third finger)	41.0	47.2
Forearm and hand (radial point to third finger)	22.0	30.1
Hand (styloid point to third finger)	10.5	13.2
Lower extremities (anterior superior iliac spine to ground)	48.0	61.7
Leg (tibial point to ground)	22.0	28.6
Foot (malleolus to ground)	2.5	4.6
Weight	13.5 kg.	19.7 kg.

The figures for the normal 6 year old female were provided by Dr. Czeslaw Zychowicz.

Table II

OBSERVATIONS OF 3 CHILDREN WITH METAPHYSEAL DYSOSTOSIS

Authors	Sex	Age	Height in cm.	Urine	NPN mg.%	Serum P mg.%	Serum Ca mg.%
Jansen[9]	M	10	95–38	normal	—	4.6	10.0
Schmidt[10]		6	86.5–25.5	normal	28	4.0	10.7
Kozlowski and Zychowicz	F	6	92–22.8	normal	23	4.8	9.6–10.7

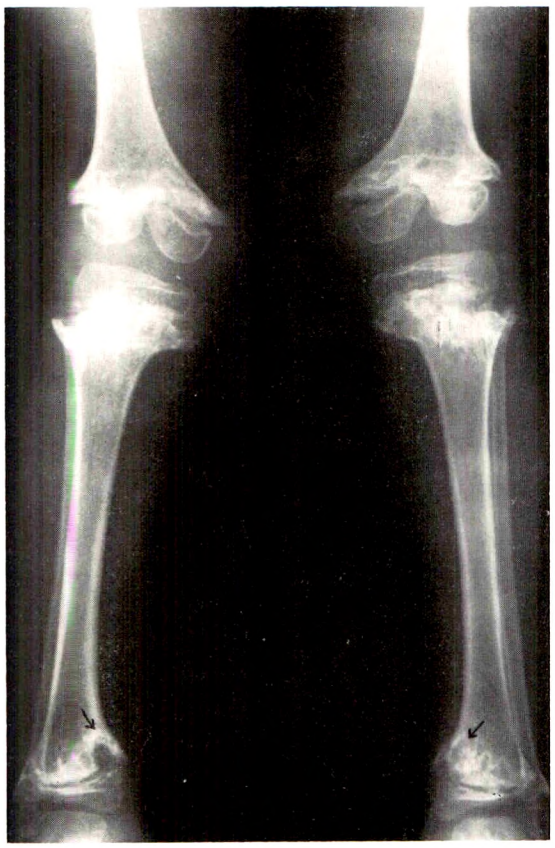


Fig. 2. The changes at the knees and ankles are shown. Both ends of both tibias are affected in a similar way. At the proximal end of each tibia, the epiphyseal ossification center is large and smooth with no evidence of marginal dysostosis. In contrast, the contiguous metaphysis is widened and irregularly ossified in a deep zone of irregular density which extends laterad and mediad well beyond the ends of the epiphyseal ossification center. There are several sharply defined radiolucent patches in the irregularly ossified metaphysis. The submetaphyseal spongiosa is deformed and irregular both in pattern and density. The remainder of the diaphysis is normal with well mineralized cortical walls and spongiosa, and the spongiosa is normal in pattern. In the distal tibial metaphyses, irregular ossification similar to that in the proximal metaphyses is present, but the depth of the irregular zone is not so great as in the proximal tibial metaphyses. In the right tibia, there is a deep punched out radiolucent patch (arrow) which has no counterpart in the other metaphyses. The ossification centers in the distal epiphyseal cartilages of the tibias are small but smooth, and there is no evidence of marginal dysostosis. In both ends of the fibulas the metaphyseal changes are similar to those in the tibias, and a small ossification center in each distal fibular epiphyseal cartilage is smooth. The distal ends of the femoral shafts are spread and irregularly ossified in the metaphyseal levels in a fashion similar, but not as marked, as in the tibial metaphyses. However, in the distal epiphyseal cartilage of each femur, there are two, rather than one, ossification centers, with the medial of these paired centers tipped caudad and mediad off the end of the shaft. The lateral counterparts of the medial centers, in contrast, either overlap on the edge of its shaft or fit into a depression in this edge.

Laboratory findings disclosed normal red and white blood cell counts and normal hemoglobin. Urinalysis: specific gravity 1.020, with normal chemical and cellular findings; normal blood and urine chromatography. The Price-Jones curve was normal. The Wassermann test was negative. The serum calcium was 9.6–10.7 mg. per cent, serum phosphorus 4.8 mg. per cent (Table II), and alkaline phosphatase activity equalled 11.3 Bodansky units. Blood nonprotein nitrogen was 23 mg. per cent and urea 32 mg. per cent. Protein: electrophoretic protein, total protein 6.0 gm. per cent; albumin 57 per cent; alpha$_1$, 8.2 per cent; alpha$_2$, 9.4 per cent; beta 10.2 per cent; gamma 15.1 per cent.

The diagnostic roentgenographic changes were confined to the metaphyses in the tubular bones of the hands and feet; the distal ends of radiuses and ulnas, both ends of the tibias and fibulas and the distal ends of the ulnas (Fig. 2, 3, and 4, *A* and *B*). In the other metaphyses—at the hips (Fig. 5), elbows, shoulders, both ends

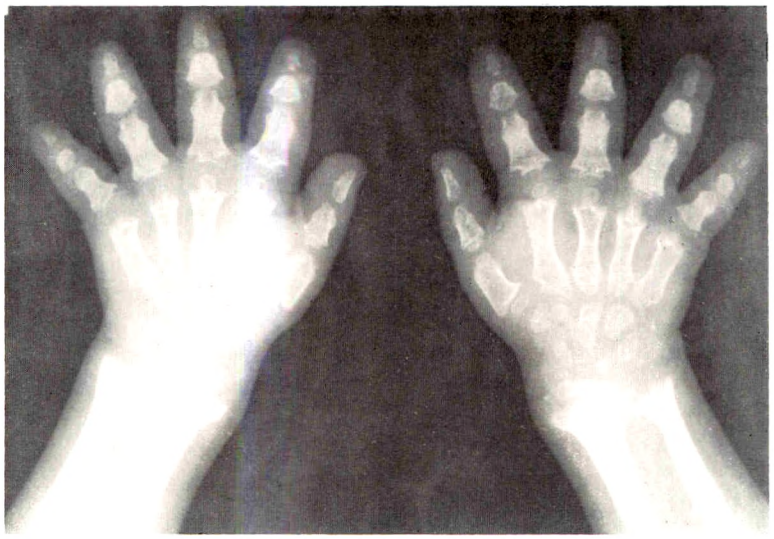


FIG. 3. In the hands and wrists, each radius and ulna are spread and cupped at their distal ends in the metaphyseal zones, but again the ossification center in the radial epiphyseal cartilage is small but smooth, and without evidence of marginal dysostosis. The round bones in the wrists are increased in number to 7, but they are all small and smooth with no evidence of marginal dysostosis. The proximal ends of the shafts of the metacarpals are pinched; the distal ends of the metacarpals have shallow transverse bands of irregular metaphyseal ossification, but here also the small ossification centers are smooth. All of the phalanges are short and broad. In all the basilar phalanges, except the first, the usual secondary epiphyseal ossification center is replaced with a transverse ridge of bone which protrudes from the central two-thirds of the base of each phalanx. The distal side of this ridge fuses imperceptibly with the main mass of its phalanx and the proximal free shallow edge of the ridge is depressed in its central segment in a concave contour which suggests the roof of an inverted pagoda. These basilar ridge deformities are due primarily to sharp oval defects in the bones, laterad and mediad at the junction of the base with its lateral and medial walls. All of the middle phalanges are extremely broad in relation to their long axes and are bell-shaped with wide proximal bases and blunt convex distal ends. The distal phalanges are all small. The distal phalanx in the second digit is the only one which has a secondary epiphyseal ossification center. The phalanges in the thumbs are only slightly dysplastic.

of the ribs and the vertebral column, slight metaphyseal irregularities were noted as well as slight deformities in the juxtametaphyseal spongiosa, but these changes were not diagnostic and they barely exceeded the limits of normal variation. The round bones in the wrists and ankles were small, but smooth, as were the epiphyseal ossification centers. The skull was long transversely, but short ventrodorsally and cephalocaudally. The calvarium was thin with retarded differentiation of the inner and outer tables and the diploic space. The ethmoidal labyrinths were markedly widened to produce a pronounced orbital hypertelorism. There was no evidence of dysostosis in the cartilaginous bones at the base of the skull. Both petrous bones were devoid of pneumatization and presented a heavy ivory-like sclerosis in contrast to the normal pneumatic structure in the paranasal bones. The nasal bones were invisible; they were either absent or nonmineralized. The pituitary fossa was normal in size, although it had a long anterior recess under the anterior clinoid processes. The dental development was normal, but the upper central incisors protruded forward in a rodent-like position to overbite on the lower teeth.

The most marked metaphyseal changes were located in both ends of both tibias and both fibulas (Fig. 2). The epiphyseal ossification centers of these bones were, in contrast, smooth and presented no signs of marginal dysostosis. Only one end of each femur was affected; the distal end, where the changes were not as marked as in the tibias. In each distal epiphyseal cartilage of the femur, there were two large ossification centers in place of the normal single center.

On the roentgenogram of the wrists and hands (Fig. 3), the changes were marked in the distal ends of the radiuses and ulnas, the metacarpals and the phalanges. The ossification center in the

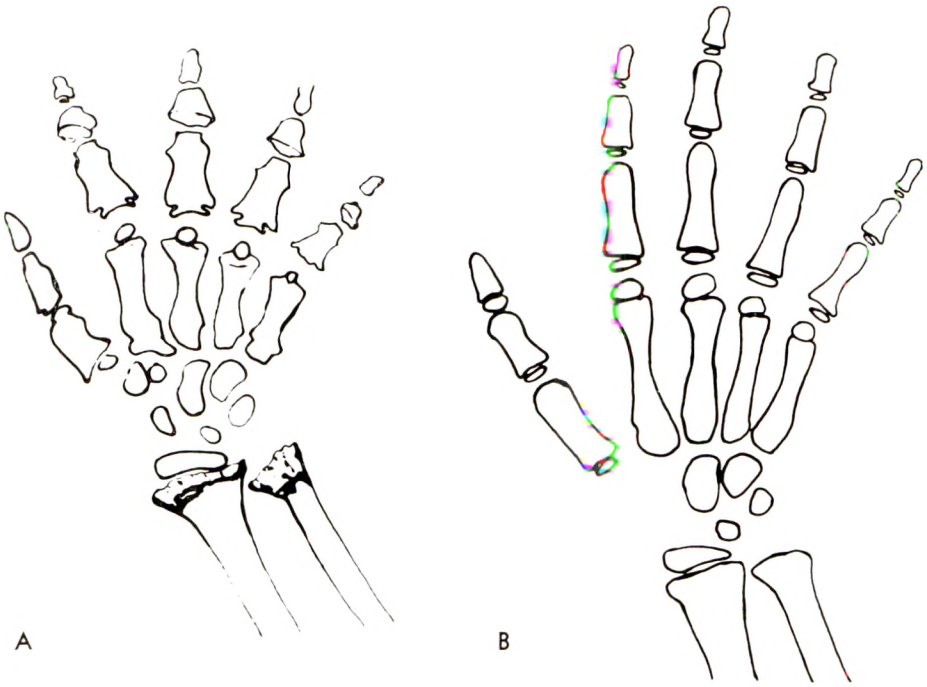


Fig. 4. In these tracings of roentgenograms of the right hand of A.K. (*A*) and the right hand of a normal American girl 6 years of age (*B*), the abnormal features described in the legend for Figure 3 are seen more clearly.

epiphyseal cartilage of each radius was small but smooth, as were the round bones in the wrists which were 7 in number. All of the bones in both hands were small and the tubular bones were large in girth in comparison with their shortened longitudinal diameters.

Tracings of one hand of the patient and of a hand of a normal American girl 6 years of age were made and these show in detail (Fig. 4, *A* and *B*) the shortening of the tubular bones, the pinching of the proximal ends of the metacarpals, and the smallness of the ossification centers in their distal epiphyseal cartilages. Also at the bases of the basilar phalanges, the usual independent ossification centers in the epiphyseal cartilages are seen to be replaced with transverse ridges which resemble a pagoda roof with upturned edges, or the shape of the base of an arrowhead, or the base of a keystone. The middle phalanges are extremely broad and short and have the shape of a bell with the base at the proximal end; there are no ossification centers in their epiphyseal cartilages. The distal phalanges are all hypoplastic, both absolutely and relative to the other phalanges.

In Figure 5, the absence of diagnostic changes

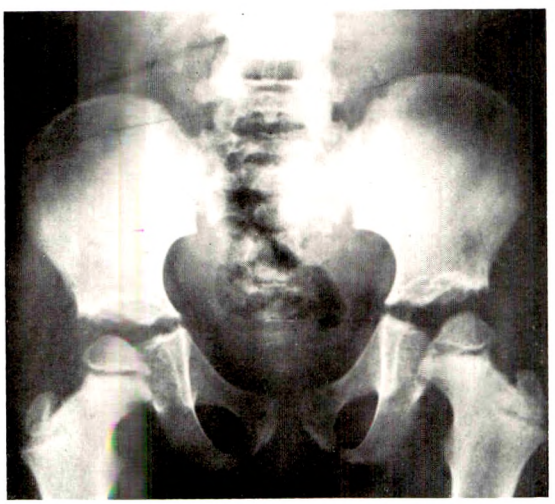


Fig. 5. The pelvis and hips of A.K. in frontal projection. There are no abnormal findings. The metaphyseal changes present in the more distal metaphyses of the bones of the legs are strikingly absent. The irregularities on the bony rim of the acetabulum are normal and these should not be considered a sign of dysostosis. The same is true for the shallow scallopings on the iliac crests. There are no changes suggestive of achondroplasia or dysplasia in pelvic or spinal bones.

in the proximal ends of the femurs, the pelvic bones, and the distal segments of the lumbar vertebras are shown.

DISCUSSION

Negative biochemical tests tend to exclude in our case renal rickets, hyperparathyroidism, hypophosphatasia, or other known metabolic diseases which can be confused with metaphyseal dysostosis.

In this case the metaphyses of the long bones are most severely affected. The diaphyses are shortened but of a normal structure. The epiphyseal ossification centers are normal in development although slightly deformed at their bases. Only the ossification centers in the distal epiphyses of the femurs are markedly deformed, and we think that this is due to abnormal mechanical stress on the epiphyses. We regard the changes in the diaphyses and epiphyses as the residuals of abnormal metaphyseal growth. It is surprising that this deformation did not cause any serious disability, but the child was described by the mother as "very alert and a good runner."

The second point to be stressed is the difference in severity of the lesions in different metaphyses. The roentgenologic findings at the shoulder, hip, and elbow joints are similar to ones found in the Schmidt type of metaphyseal dysostosis, but the changes in all the other big joints would warrant the diagnosis of the Jansen type of disease. The roentgenologic image in the metacarpals, metatarsals, and the phalanges of the fingers and toes are not so severe as in the Jansen type, but much more severe than in the Schmidt variety, in which the bones of the hands and feet may be normal. There are some further roentgenologic observations in our case which are worth stressing: the skull was affected, and irregular trabeculation was present not only in the metaphyseal parts of the long bones but also in the carpal and tarsal bones as well. This indicates a generalized disturbance of endochondral ossification.

Microscopic examination of a fragment from the lower end of the tibia in a Jansen type case showed cartilage cells of irregular size and arrangement with a distorted zone of provisional calcification and isolated masses of bone within the substance of the cartilage. No microscopic examinations of carpal or tarsal bones were described.

A detailed study of the two varieties of metaphyseal dysostosis, the Jansen type and Schmidt type, and their comparison with our case disclose some differences which make it difficult to classify our case. We cannot explain satisfactorily the difference in the severity of disturbances in the growth in the different individual metaphyses. Jansen believed that the abnormality in his case was due to intrauterine trauma. We think that it is now more reasonable to attribute the differences in roentgenologic and morphologic appearances to various expressions of the same or similar genes responsible for the disease. It is possible that these modifications may also be caused by the sex chromosomes; however, it is possible that the difference in the severity of the disease depends on the stage of embryologic development in which the causal factor, inherited or environmental, started to operate.

In view of the constant difficulties in classification of endochondral dysplasias, incomplete and intermediate forms which suggest transitional forms of cartilaginous dysplasias have long been noted. We think that Weil's classification of metaphyseal dysostosis in the Schmidt and the Jansen type is acceptable for the clinic, but there are intermediate cases resembling more either one or the other variety, which are difficult to classify. Our case is an example of this. The involvement of the skull and the character of the trabeculation of the long and short bones may cause further confusion. As long as no causal factors of the endochondral dysplasias can be demonstrated, the careful description of the peculiarities of single cases is useful. The classification of similar cases into clinical

and morphologic groups seems reasonable, as it might be of value when more is known of their pathogenesis and natural course.

SUMMARY

Metaphyseal dysostosis in a female 6 years of age is described. Roentgenograms showed disturbances of ossification and growth in the metaphyses and contiguous irregular trabeculation in several bones. The metaphyses were affected in markedly different degrees in different bones. The skull and spine were only slightly affected.

The findings in this patient overlap both the Jansen and the Schmidt types of metaphyseal dysostosis. This case should be considered the intermediate type or link between the severe generalized disease of Jansen and the milder type of Schmidt.

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GENERALIZED SKELETAL DYSPLASIA WITH MULTIPLE ANOMALIES*

A NOTE ON PYLE'S DISEASE

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SEVERAL diseases with an increase in density and/or thickness of the skull have been described in children. Craniometaphyseal dysplasia (Pyle's disease),[11,15] fibrous dysplasia, idiopathic hypercalcemia,[6] idiopathic hyperphosphatasia,[3,18] and osteopetrosis are some diseases with these manifestations. The general picture of the case being reported in this paper does not completely fit into the description of any of the above or other diseases reviewed by the author.

REPORT OF A CASE

D.W. (Hosp. #20-39-01) is a 10 year old white male born on October 9, 1951. He was the product of a normal full term pregnancy and delivery. The birth weight was 2.9 kg. (6 lb., 7 oz.). Unusual facial appearance, microstomia, cleft palate and deformities of hands and feet were noted at birth.

Family History. The father was 27 and the mother was 26 years of age when the patient was born. They both were in good health. He has an older female sibling who has a cardiac murmur of unknown etiology but is otherwise normal. Roentgenographic examinations of the skeleton of the parents and sister were unremarkable. The only relative known to have been abnormal was the mother's maternal aunt who had cleft palate, a "soft head" and according to one observer looked very much like this patient. She died at 10 years of age. The cause of death is not known.

Past History. Growth and development have been slow. He recognized his mother at about 5 months of age. At 12 months, he began to creep; at 14 months, he pulled himself up; and at 15 months, he could walk around objects. He could not walk without support until about 2½ to 3 years of age, probably due to his hip disease. He could say "mama" and "dada" when he was about 12 months old, but was slow in his speech development. At his first visit to the Medical Center he was 2 years old. His weight was 10.9 kg. (23 lb., 13 oz.) (3 percentile) and his height was 76.25 cm. (30½ inches) (3 percentile). The head circumference was 48 cm. (19¼ inches) (normal). The anterior fontanel was wide open measuring 7.5 cm. in width. A 3×1 cm. opening in the posterior hard palate was present. He had bilateral hip dislocation. Reduction of the dislocation was achieved and a cast was applied. Stomatoplasty for microstomia and palatoplasty were performed at 8½ years of age. He has had repeated bouts of middle ear infection. Dental examination at 9 years of age showed multiple impacted and missing teeth with only 10 erupted teeth present. Speech evaluation revealed severe articulation difficulty related to cleft palate, microstomia, a small oral cavity with a high anteroposterior vault, an apparently short and immobile velum, and a flat nose. The hearing test indicated bilateral hearing loss of conductive type. He developed suppurative epididymitis on the right side at 9 years of age and underwent an operation for this.

Physical Examination. At 10 years of age the patient's weight is 21.4 kg. (47 lb.) (below 3 percentile) and the height is 121.25 cm. (48½ inches) (below 3 percentile). He has a prominent head, protruding forehead and occipital region, receding eyes, hypertelorism, small and flat nose and a small oronasal fistula (remaining following operation) (Fig. 1). A chest deformity consisting mainly of a moderate degree of pectus excavatum is present. The heart and lungs are unremarkable. The blood pressure is 100/80 mm. Hg. No hepatosplenomegaly is present. The extension of the upper arms at shoulder is mildly limited. The elbow extension is bilaterally markedly limited (120° to 130°). Both wrists are in pronation and cannot be supinated. The deformities of the hands and feet are shown in Figure 2. Some tightness of both hips is

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present. The knee extension is complete but there is some limitation of flexion.

He is mentally somewhat retarded (IQ 74); the hearing problem is partially responsible for this retardation.

Laboratory Findings. Blood: Hemoglobin, 13.7 gm. per cent; white blood cells, 7,600 with a normal differential count; sodium, 140 mEq./L.; K, 3.5 mEq./L.; Cl, 102 mEq./L.; CO_2, 28.22 mEq./L.; calcium, 10.0 mg. per cent; phosphorus, 3.8 mg. per cent; alkaline phosphatase, 5.5 Bessie-Lowry units; urea nitrogen, 11 mg. per cent; and fasting blood sugar, 90 mg. per cent. *Urine:* Routine urinalysis was negative. The urinary aminoacid pattern was evaluated in Dr. H. N. Kirkman's Laboratory and reported as not being abnormal.



Fig. 1. The patient at 9 years of age. Photographs showing the small trunk and extremities in comparison with the prominent skull. He has hypertelorism, receding eyes, microstomia, and deformities of the extremities.

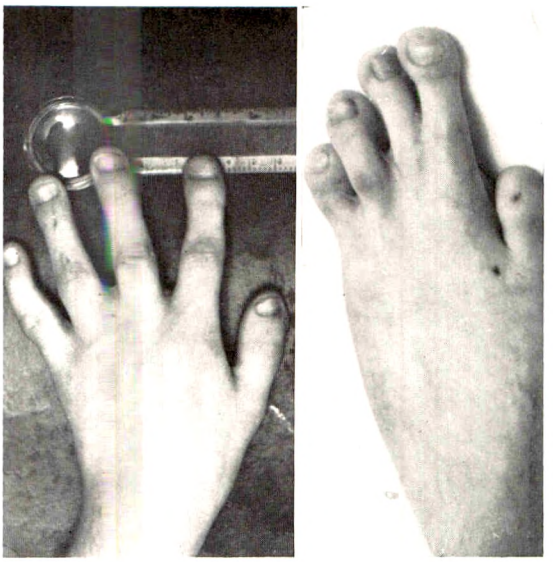


Fig. 2. Photographs showing the abnormally wide distal phalanges of the hand and foot, and a very short first toe with hypoplasia of the nail.

Roentgenographic Findings. Skeleton roentgenograms from the age of 2 years to 9 years are available for evaluation.

At 2 years of age the skull was dense and had an abnormal shape with a large occipital region. The density was most marked at the base. The anterior fontanel was widely open (Fig. 3*A*) and a wide, noncalcified segment was present between the two halves of the frontal bone. The sella turcica was very wide and had an irregular floor. Roentgenograms taken at 9 years of age showed persistence of increase in bone density at the base of the skull. A marked decrease in the size of the open fontanel had taken place, but this still remained open (Fig. 3, *B* and *C*). The diploic space was not visible. Multiple sutural bones were still present in the occipital region. The posterior fossa was deep. The supraorbital ridge was very prominent. The foramen magnum was relatively small and had irregular anterior and posterior borders. The mastoids and paranasal sinuses showed no roentgenographic evidence of pneumatization. The ossicles in the middle ear were densely visualized. The optic foramina each measured 3 mm.; the vertebrae were slightly to moderately denser than normal. The neural arches of several lumbar and sacral vertebrae were not completely fused (Fig. 4). A moderate to marked degree of pectus excavatum was present (Fig. 5, *A* and *B*). The ribs were unremarkable except for de-

formities related to the pectus excavatum. There was a generalized lack of normal modeling of long bones, most marked in the phalanges. The radial heads were dislocated posteriorly (Fig. 6). The proximal and distal ends of the radii and ulnae were abnormally formed (Fig. 6 and 7, *A* and *B*). On roentgenograms taken at $2\frac{1}{2}$ years of age, only one ossification center was present in the right wrist (Fig. 7*A*) and none on the left side. On roentgenograms taken at 9

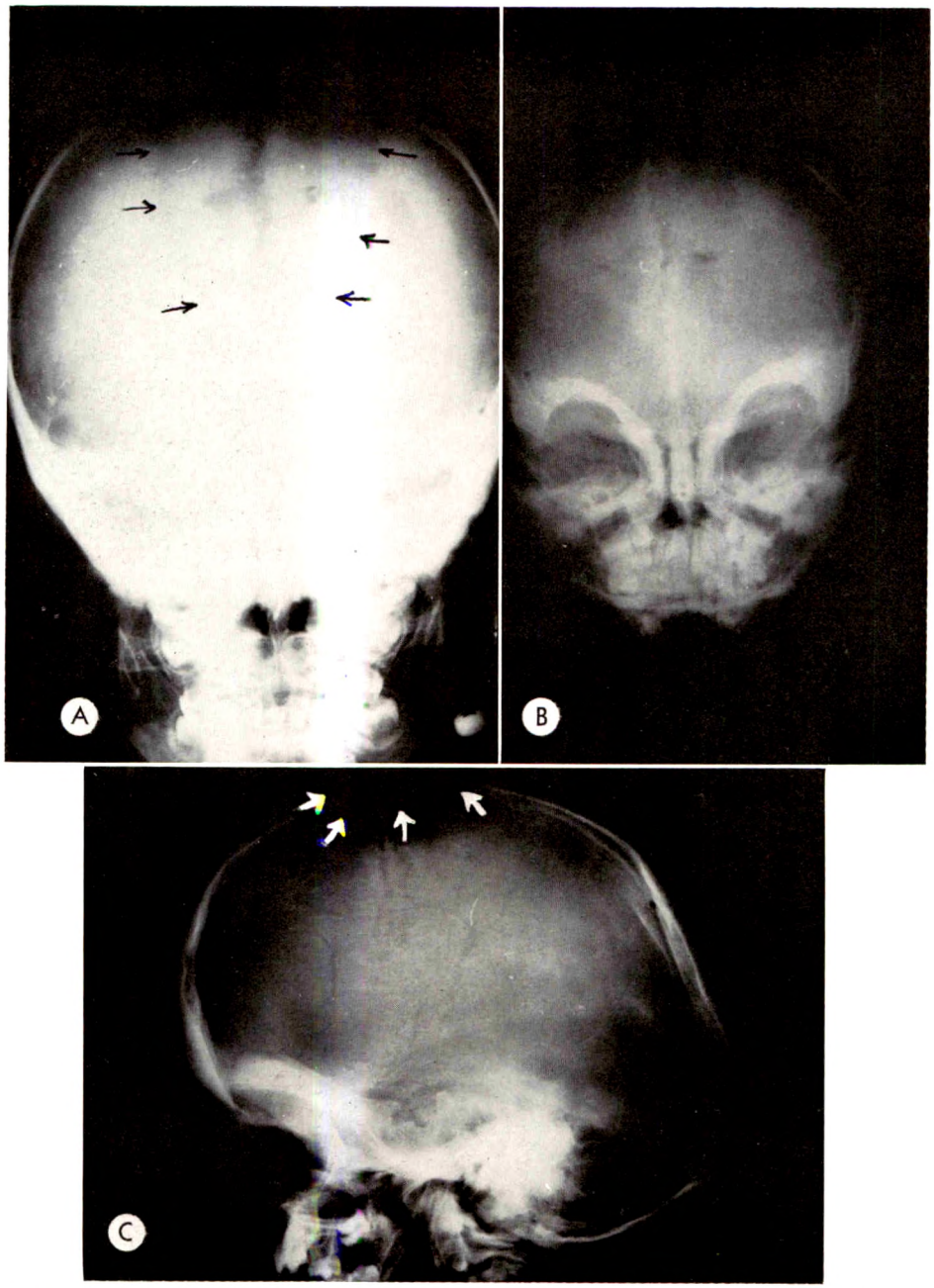


FIG. 3. (*A*) Roentgenogram of the skull at 2 years of age shows the dense bone and very wide anterior fontanel and metopic suture. (*B* and *C*) Roentgenograms showing the dense bone, in particular at the base, a deep upper occipital region, an open anterior fontanel, heavy supraorbital ridges and relatively small facial bones at 9 years of age.

years of age, 5 ossification centers were present on each side. The carpal bones were abnormally formed and irregularly shaped with a marked delay in maturation (Fig. 7*B*). The metacarpals and phalanges were grossly underconstricted. The middle phalanx of the fifth finger was relatively shorter laterally, resulting in curved fingers. An early fusion of an abnormally elongated secondary ossification center of the distal phalanx of the thumb was noted at 6 years of age. A notch at the site of fusion was visible at 9 years of age. The ischiopubic bones appeared wider than normal. Bilateral hip dislocation was present at $2\frac{1}{2}$ years of age (Fig. 8). Roentgen examination at 9 years of age revealed bilateral coxa plana, coxa magna and irregular acetabular roofs. The femora had medial bowing with a thick cortex, mostly medially (Fig. 9*A*). The patellae were calcified. The epiphyses at the knees were not remarkable, except for coarse trabeculae. The tibiae had a wide "S" shaped appearance (Fig. 9*B*). The metaphyseal regions were not unusually wide. The distal epiphyses of the tibiae and fibulae were large. The feet were markedly abnormal (Fig. 10, *A* and *B*). At $2\frac{1}{2}$ years of age they were pronated. The calcanea, tali, and cuboids were the only three calcified tarsal bones at that time and these were abnormally formed. At 9 years of age, the cuboids were relatively large in comparison with the naviculars, which had two irregular ossification centers each. The cuneiforms were irregular and abnormal in shape. The second and fourth metatarsals had an irregular and elongated proximal accessory ossification center. Bilaterally, the first metatarsals and the phalanges of the first toes were short, wide and very ab-



Fig. 4. Roentegnogram shows the dense vertebrae with a lack of complete closure of some of the neural arches.

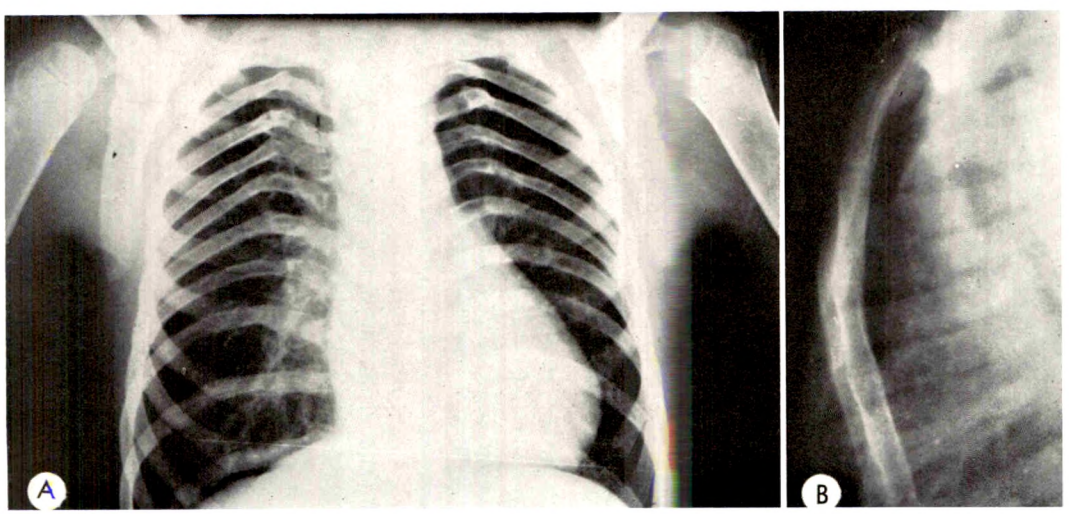


Fig. 5. (*A* and *B*) Roentgenograms show pectus excavatum, thick scapulae and underconstriction of humeri.

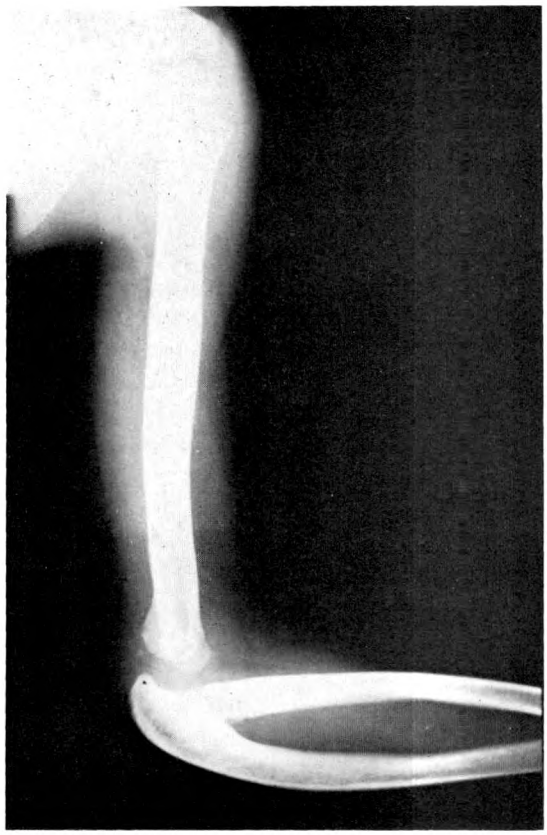


FIG. 6. Roentgenogram shows abnormal modeling of humerus, radius and ulna, and dislocation of the head of radius.

normally formed. The other metatarsals had a long "S" shaped curve. The toes were relatively long and drumstick-shaped. On both sides, the distal phalanx of the second toes appeared to have formed from two incompletely fused centers.

DISCUSSION

Virchow[19] in 1864 described the condition known as leontiasis ossea, a hyperostosis affecting the facial and cranial bones. It was later suggested[8] that the term "leontiasis ossea" be used for any hyper-

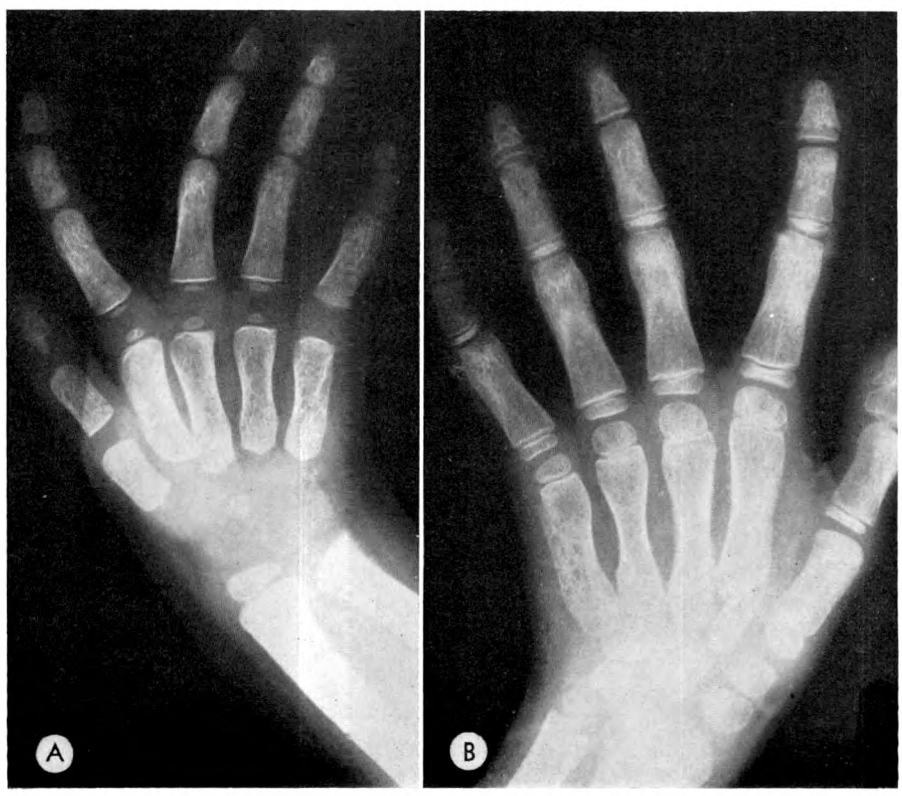


FIG. 7. (*A*) Roentgenogram taken at $2\frac{1}{2}$ years of age reveals an abnormal modeling of the bones of the hand. Note the long secondary ossification center of the distal phalanx of the thumb. (*B*) Roentgenogram shows the large distal epiphysis of the radius, and abnormal shape of the carpal bones, metacarpals and phalanges at 9 years of age.



Fig. 8. Roentgenogram at $2\frac{1}{2}$ years of age shows dislocation of hips, lack of appearance of secondary ossification centers of femoral heads and a wide ischium.

ostosis of unknown origin and of rare occurrence chiefly affecting the skull. The diseases in which sclerosis of the skull in children is seen are: Paget's disease,[4] osteopetrosis, hyperphosphatasia,[3,18] fibrous dysplasia,[10,19] and craniometaphyseal dysplasia,[5,11,13,15,16] but there are other conditions with dense skull which are not classifiable.[2,17] Our case could be considered to be one of these.

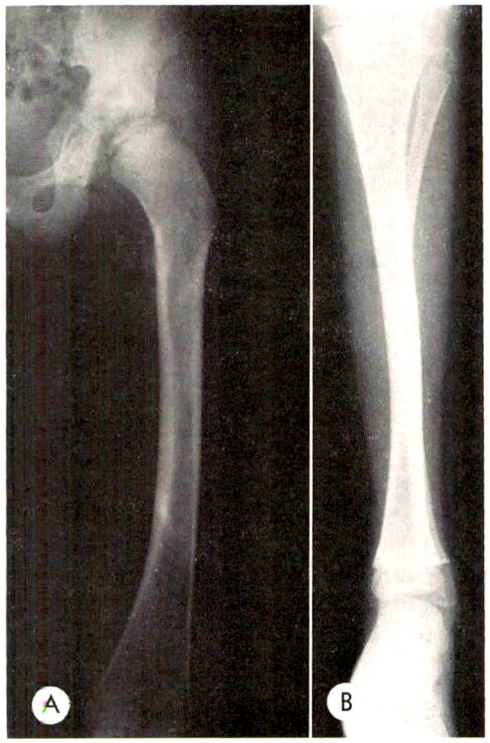


Fig. 9. (*A*) Roentgenogram shows a flat femoral head, wide femoral neck, and medial bowing of the femur at 9 years of age. (*B*) Roentgenogram shows a wide "S" shaped appearance of the tibia and the fibula is medially curved.

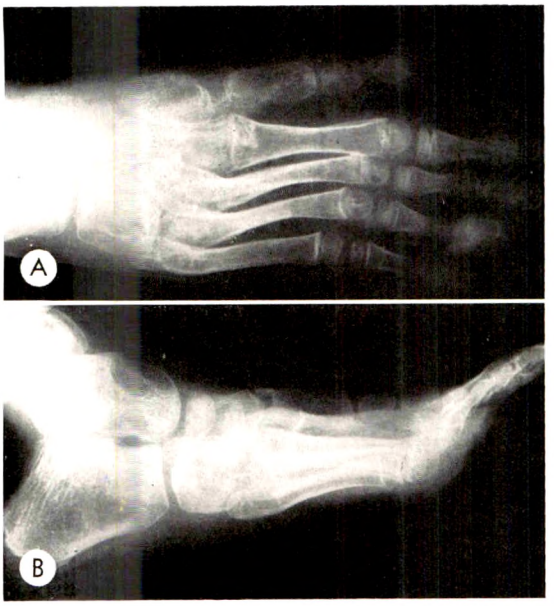


Fig. 10. (*A* and *B*) Roentgenograms showing an abnormal shape of the tarsal bones. The first metatarsal and phalanges of the great toe are short, wide and grossly abnormal. The remaining metatarsals are relatively long.

The skull findings of this patient have some resemblance to those of Pyle's disease.[15] Unaware of Pyle's description of this new disease 2 years earlier, Cohn[5] reported a case with excessive sponginess of the skeleton. The findings as described in his paper were typical of craniometaphyseal dysplasia and included were roentgenographic findings of the skull which were not known to Pyle.[15] Cohn described a large posterior part of the skull, the thickened bone and the small sella turcica in his patient. This case with good demonstration of skull abnormalities of this entity has not been quoted and commented upon in the English literature.[1,7,9,11,12,13,14,16] To give Cohn recognition for his excellent and complete description of this disease, the author suggests that the entity be named Pyle-Cohn's disease.

A review of the cases with craniometa-

physeal dysplasia (Pyle-Cohn's disease) fails to show a report of wide fontanel and sutures and the delay in their closure that was seen in the case presented here. The typical wide metaphysis seen in craniometaphyseal dysplasia is absent in this patient, though a degree of abnormality of modeling is present. In addition, physical retardation and abnormalities of chest, wrists, hands and feet described in our patient have not been reported in this disease. The slight limitation of extension of the elbow in craniometaphyseal dysplasia is presumably due to the encroachment on the olecranon fossa by metaphyseal widening,[11] but congenital dislocation of the elbows is not part of the clinical picture of this disease.

The wide fontanel and sutures, incomplete closure of neural arches of the vertebrae and dentition abnormalities may suggest a diagnosis of cleidocranial dysostosis, but this is excluded by the lack of other typical findings of the disease and by the presence of other anomalies that are not a part of this disorder.

The facial appearance may suggest Cruzon's craniofacial dysostosis; however, the absence of premature synostosis of coronal sutures, prognatism of the mandible, and the presence of deep orbits leaves such a diagnosis without support.

Osteopetrosis should not be considered a possibility in this patient due to the absence of an increase in the density of the long bones, anemia, and because of the presence of other anomalies.

SUMMARY

1. An unusual generalized osseous dysplasia with multiple anomalies is reported. These anomalies include, dwarfism, deformity and increase in density of skull, delayed closure of anterior fontanel, dislocation of hips and elbows, and deformities of chest, hands, and feet.

2. The differential diagnosis from other entities is considered.

3. The resemblance of cranial manifestations in this case to those of craniometaphyseal dysplasia is discussed, and the suggestion is made that this latter entity be named Pyle-Cohn's disease in recognition of Cohn's first complete description of this condition, including the cranial manifestations.

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ROENTGEN MANIFESTATION OF THE ALDRICH SYNDROME*

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IN 1954, Aldrich *et al.*[1] reported a sex-linked recessive condition in an infant and commented on several male members of this family. The illness was characterized by congenital thrombocytopenia, bloody diarrhea, eczema, and recurrent otitis media. Severe episodes of bronchopneumonia and pansinusitis as well as other manifestations of infection were present. Aldrich was able to trace the pedigree of this family for 6 generations.

The condition is transmitted via a familial sex-linked recessive gene attached to the X chromosome. It is carried by the female and manifested only in male infants, much like hemophilia. Since the original presentation of Aldrich *et al.*, at least 20 cases have been described in the literature.[2–9,11] The reported cases died early, either due to infection or hemorrhage. Many of the studied cases showed a lack of natural isoagglutinins against heterospecific blood group antigens.[8]

The previously reported cases, as well as our series, have features of roentgenographic importance. Recurrent pneumonias in a patient with eczema and thrombocytopenia plus repeated otitis suggest the diagnosis. The positive family history is the confirmatory feature.

We have had an opportunity to study 5 cases of the Aldrich syndrome at Babies Hospital. Three of these cases were in a single Negro family and are the first 3 cases of this report. Two of these 3 cases have been previously reported[3] (Fig. 1). The other 2 cases are in white male infants who demonstrate the typical features described by Aldrich.

REPORT OF CASES

CASE I. T.S. (105-70-14) developed eczema at at the age of 3 weeks covering the trunk and extremities. Nine days prior to his first admission to Babies Hospital, at the age of 7 weeks, he had numerous blood-streaked stools. On admission he had an eczema, hepatosplenomegaly, anemia, a platelet count of 82,000 per cubic millimeter, and a white blood cell count of 9,400. The bleeding time was prolonged and the clot retraction was poor at the end of 24 hours.

During this admission, the platelet counts ranged from 25,000 to 88,000 per cubic millimeter. Subsequently, this patient had 24 hospital admissions for problems related to his illness including bloody diarrhea, nonspecific pneumonitis (Fig. 2 and 3), epistaxis, hematemesis, skin infections, sinusitis and mastoiditis (Fig. 4 and 5). Figure 6 is a graphic representation of the clinical course over the first 5 years of life.

This patient also had migratory joint pains and effusions (Fig. 7, *A–C*), a palpable thyroid, Salmonella enteritis, persistent anemia, and a peripheral eosinophilia. There was no increased number of megakaryocytes in the bone marrow as one might expect to find in idiopathic thrombocytopenic purpura.

Currently, this patient is doing well and is $10\frac{1}{2}$ years old. All the patients reported in the literature have died by $5\frac{1}{2}$ years except one who died at 7 years. This is the longest survivor and such longevity is very unusual for this disease.

CASE II. G.T. (120-91-57), a first cousin of the previously described patient, had, during the $6\frac{1}{2}$ years of his life, 24 hospital admissions (Fig. 8). In early infancy he developed eczema, thrombocytopenia, bloody stools, and chronic otitis media. During his many hospital admissions he had repeated episodes of bronchopneumonia (Fig. 9 and 10), bilateral joint

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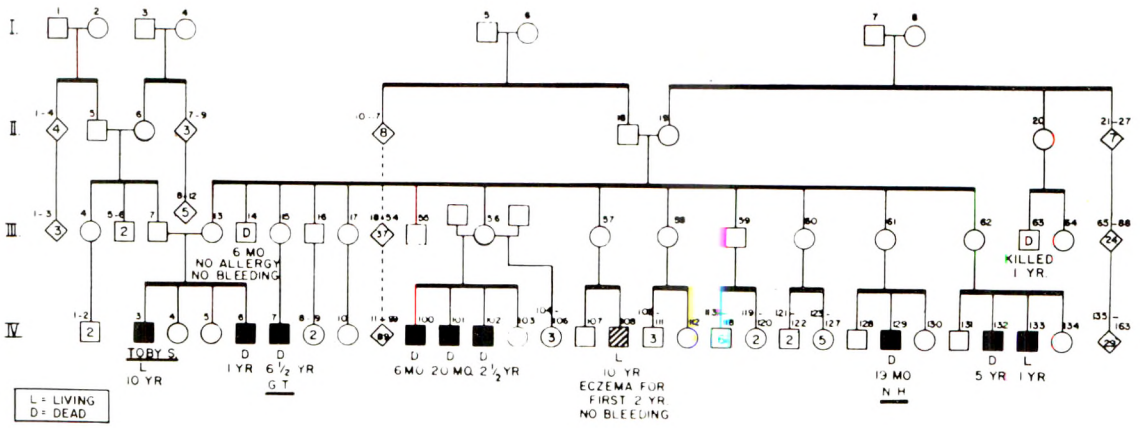


FIG. 1. The family pedigree of the first three cases, T. S., G. T., N. H., and the other first cousins whom we have not seen.

effusions which spontaneously subsided, Salmonella enteritis, and epistaxis. He also developed an enlarged thyroid while on Mullsoy feedings. During the course of his many admissions he had two episodes of meningitis, one pneumococcus type 4 and the other, hemophilus influenza type B.

On his last admission, at $6\frac{1}{2}$ years, he had hepatosplenomegaly, bleeding from the gastrointestinal tract, urinary tract, nose, and clinical and roentgenographic evidence of pansinusitis and right mastoiditis (Fig. 11 and 12). Terminally, the patient became icteric, bled from all orifices, and did not respond to repeated transfusions. Congestive heart failure and a

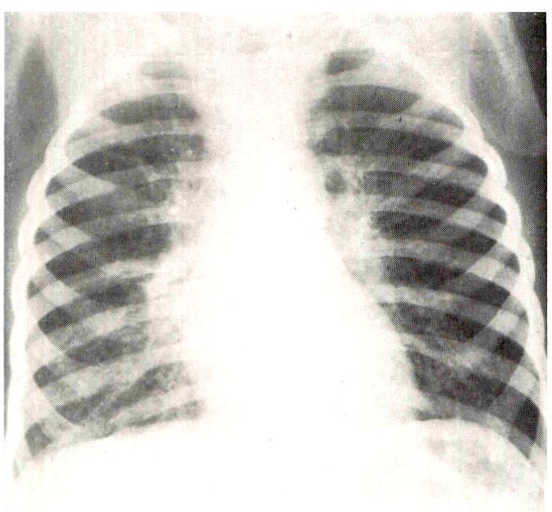


FIG. 2. Case 1. One example of the many diffuse bronchopneumonias the patient had during the first 5 years of life.

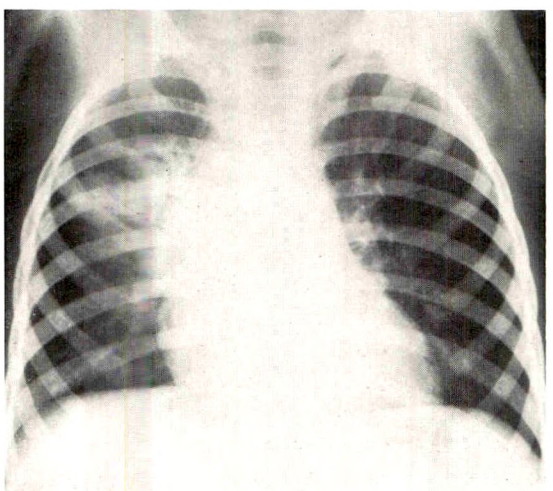


FIG. 3. Case L. Localized pneumonia at the age of 6.



FIG. 4. Case 1. Left maxillary sinusitis; one example of the many attacks.

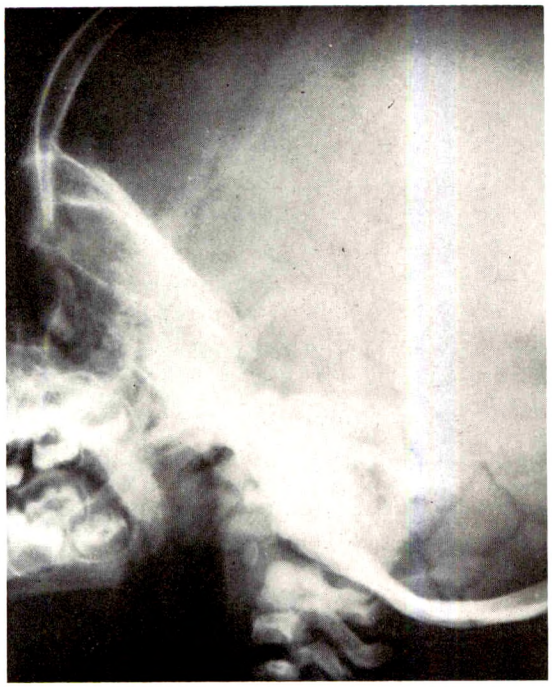
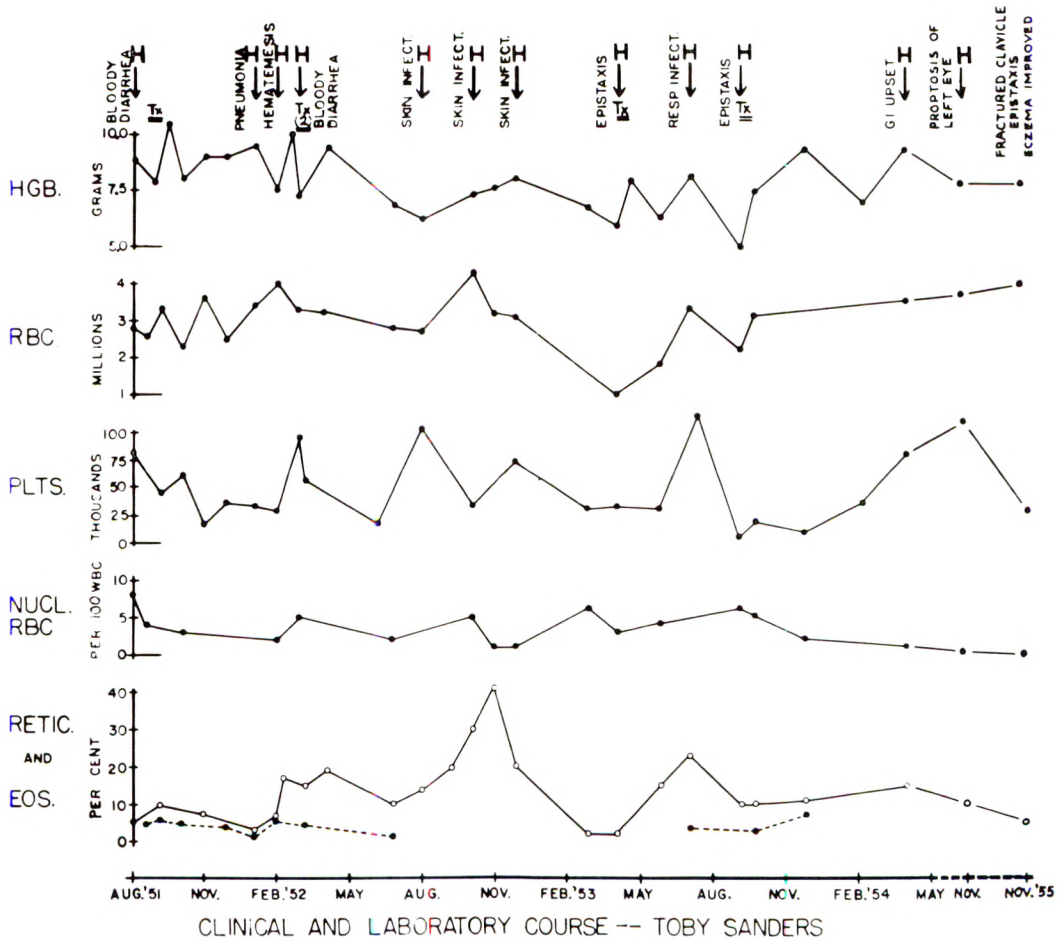


FIG. 5. Case 1. A lateral roentgenogram of the skul showing an absence of nasopharyngeal soft tissue prominence in the region of the adenoids. This has been a constant finding, even during severe sinus infections.

gallop rhythm developed and he expired 6 weeks after admission. Postmortem blood culture showed hemolytic streptococci. There were multiple acute gastric ulcers and petechiae in all layers of the heart, both kidneys, both lungs, and the intestinal mucosa. Mycotic abscesses were present in the liver with intracellular bile

FIG. 6. Case 1. The chart shows the clinical course along the top line and the corresponding laboratory data on the side of the graph. This is a fairly typical course, although several of the patients have had exacerbations more frequently.



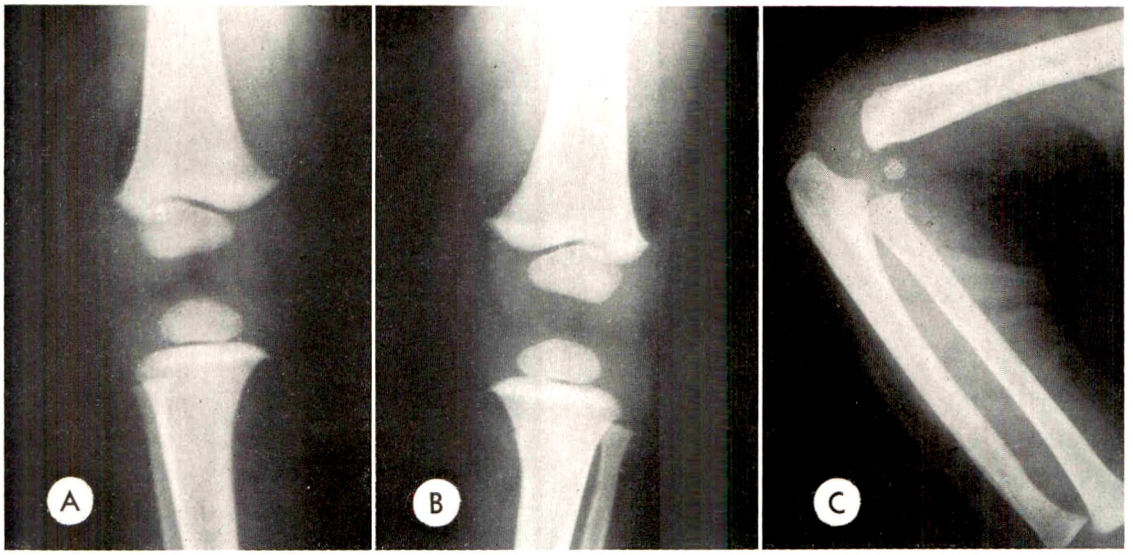


Fig. 7. Case I. (*A* and *B*) Roentgenograms of the knees and (*C*) of the right elbow at different times showing the joint effusions, which were transitory in the left knee and the right elbow.

stasis. The immediate cause of death was felt to be terminal hemorrhagic diathesis with septicemia.

Case III. N.H. (25-88-87), another first cousin, developed eczema, thrombocytopenia, and ear infections during his first month of life (Fig. 13). His first admission to Babies Hospital was at the age of 5 months for repair of a left inguinal hernia. The skin showed a severe eczematoid rash and a thick gray material was present in both ear and nasal canals. He had a hemoglobin of 10.5 gm. per cent, a platelet count of 111,000 per cubic millimeter, and a white blood cell count of 16,300 with a shift to the left of the granulocytes. He was readmitted at the ages of 7 months, 1 year, and 14 months for exacerbations of eczema, pyodermia, and pneumonia (Fig. 14, *A–F*). The laboratory findings during the final admission showed a hemoglobin of 7.6 gm. per cent, a platelet count of 22,000 per cubic millimeter, a white blood cell count of 30,000 with a shift to the left of the granulocytes and 41 per cent eosinophils. Roentgenograms of the paranasal sinuses



Fig. 8. Case II. G. T. at 9 months of age showing chronic eczema, the large axillary and supraclavicular lymph nodes, and the wasted extremities. There was no adenoid tissue seen in the nasopharynx on roentgenographic study at this time.

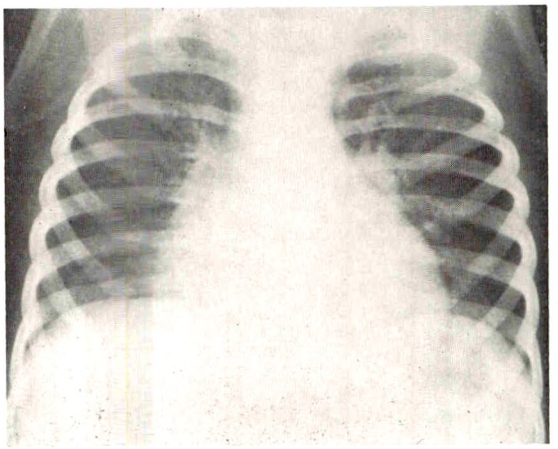


Fig. 9. Case II. Frontal chest roentgenogram showing one of the many episodes of diffuse bronchopneumonia—this at age of 3 years.

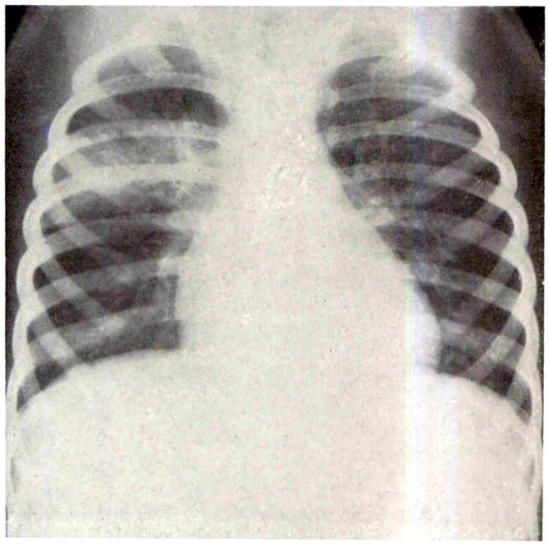


FIG. 10. Case II. Frontal chest roentgenogram showing localized and diffuse pneumonia at $3\frac{1}{2}$ years.

showed pansinusitis (Fig. 15). He improved initially during this admission but had repeated infections of his skin, sinuses, and lungs. On one occasion a bone marrow aspirate had normal cellular components but was devoid of megakaryocytes. He received repeated transfusions in an attempt to correct a refractory anemia. At



FIG. 11. Case II. Roentgenogram of the paranasal sinuses at 5 years and 3 months showing diffuse opacification of the maxillary and ethmoid sinuses.



FIG. 12. Case II. A lateral roentgenogram of the face showing no soft tissue swelling in the adenoid region even though there is obvious maxillary sinus involvement.

18 months of age, after 4 months hospitalization, he suddenly developed a spastic quadriplegia, coma, and rigidity with no localizing signs. He expired at the age of 19 months.

The autopsy demonstrated the cause of death to be a massive subdural hematoma. There was also evidence of chronic and active infection in the lungs and middle ears, focal embolic interstitial nephritis, and infiltration in the portal spaces of the liver. There were multiple duodenal ulcers, a finding unusual in children, felt possibly to be secondary to malnutrition. No morphologic feature was found which could explain a lowered resistance to infection.



FIG. 13. Case III. N. H. at 18 months showing severe eczema and epistaxis as well as hemorrhagic excoriations.

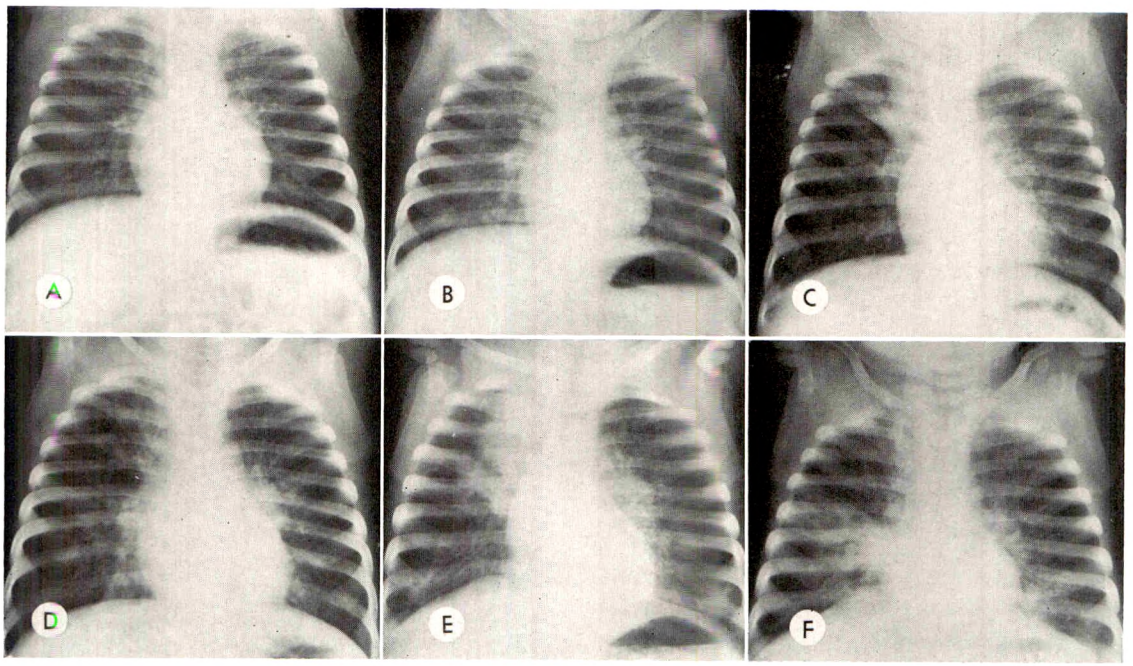


FIG. 14. Case III. Frontal roentgenograms of the chest over a 9 month period showing the severe and repeated pneumonias to which these patients are subject. (*A*) August 29, 1956. (*B*) October 15, 1956. (*C*) February 5, 1957. (*D*) March 9, 1957. (*E*) April 23, 1957. (*F*) May 27, 1957.

CASE IV. J.M. (149-39-04) in infancy developed eczema, an enlarged liver, multiple ear infections, and gastrointestinal bleeding. His platelet counts were usually under 60,000 per cubic millimeter, and he has a low titer of isoagglutinins. He is currently doing well. His brother died with the disease at the age of $4\frac{1}{2}$ years. He, like our patient, manifested the disease early but in a fairly mild form. He did reasonably well for 4 years but then rapidly became worse and expired from a cerebrovascular accident. He had eczema, otitis media, gastrointestinal bleeding, hematuria, petechiae, and a platelet count of 12,000 at the time of death.

CASE V. J.O.B. (154-99-93) was a foundling, and no antecedent history is known. At 15 months he had eczema, foul-smelling stools, and a history of multiple ear infections and recurrent pneumonia. He was first seen at Babies Hospital at 23 months of age because of failure to thrive, a skin rash, and diarrhea. Examination at this time revealed eczema and bilateral otitis externa. Roentgenograms disclosed a density in the right upper lobe interpreted as atelectasis, and retarded skeletal maturation. His platelet count was noted to be 83,000 per cubic millimeter, and he had several positive

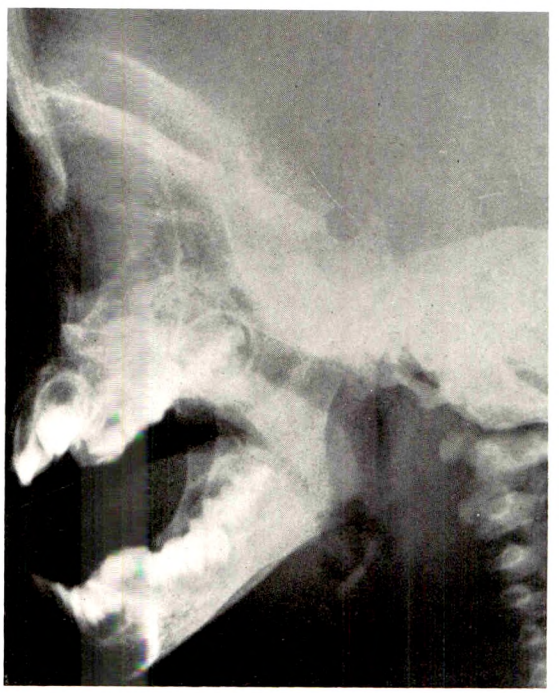


FIG. 15. Case III. Roentgenogram of the paranasal sinuses in lateral view showing the clouding of the maxillary and ethmoid sinuses, and the absence of any swelling in the adenoid region of the nasopharynx.

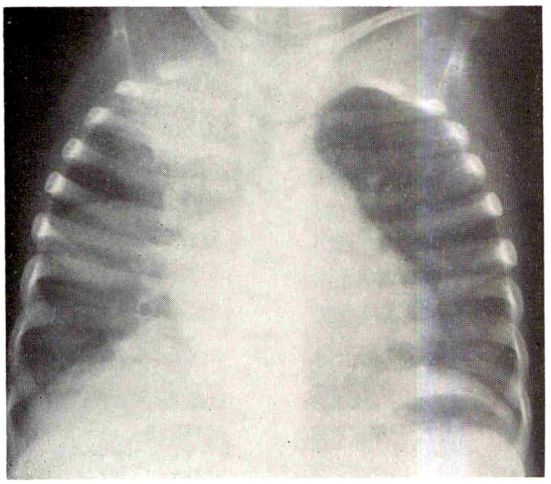


FIG. 16. Case v. Frontal chest roentgenogram showing right upper lobe atelectasis and pneumonia and right middle lobe pneumonia. The latter finding gradually cleared, while the former persisted.

guaiac stools. During the course of his hospitalization, it was noted that his platelets varied from 12,000 to 80,000 with occasional counts of 100,000 per cubic millimeter. He demonstrated a lack of isoagglutinins and a persistent eosinophilia. During hospitalization, the right upper lobe density persisted and he developed a middle lobe pneumonia (Fig. 16) and a pleural effusion, which gradually cleared. The child remained in the hospital until his death at the age of 27 months, having pursued a steady downhill course with frequent febrile episodes, frequent tarry stools, and gross rectal bleeding on many occasions.

DISCUSSION

The disease manifested by these patients is typical of the syndrome first described by Aldrich *et al.*[1] The mother of Case 1 has a remarkable memory and was able to supply us with the information necessary to construct the family pedigree (Fig. 1). It can be noted that in addition to the 3 male first cousins reported here there are 5 other male first cousins in this family whom we have not seen but who do or did have the disease. The mothers of all these patients are sisters and none of their female offspring are involved. Case IV, a male, had a brother with identical symptoms, although the nature of the genetic transmission could not be established.

We wish to emphasize the severe and recurring pneumonias that all of our patients have shown, as well as the infections of the paranasal and mastoid sinuses. The lack of any lymphoid response in the adenoid region of the nasopharynx is of interest because these children are in an age group where this tissue is normally prominent and increases in response to infection in the area. They do have an adequate lymphoid response to infection elsewhere in the body and their gamma globulin levels are normal or increased. This seeming paradox, noted even during attacks of pansinusitis, has not been reported before although patients with generalized lymphoid deficiency such as agammaglobulinemia do show a lack of adenoid tissue. Two of our patients had joint pains and joint effusions, which subsided without specific therapy and we postulate that they were serous effusions related to the hypersensitivity state. These same 2 patients showed persistent thyroid enlargement after being on Mullsoy feedings.[10] All of our patients had enlarged livers. One is surviving at $10\frac{1}{2}$ years and is the oldest recorded patient with this disease.

The radiologist should be familiar with this condition because, while the roentgenographic changes are not specific, they are typical enough so that in combination with the clinical features the disease can be recognized.

SUMMARY

The Aldrich syndrome is a sex-linked recessive disease characterized by congenital thrombocytopenia, eczema, bloody diarrhea, and anemia. Pyodermia, otitis, and pulmonary infections are common manifestations of the disease.

Five cases are reported with a genetic representation of 3 cases belonging to one family.

Roentgenographic findings representing the consistently positive features are described—recurrent severe bronchopneumonias, recurrent mastoiditis, and an apparent

absence of lymphoid tissue in the nasopharynx.

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OSTEOCHONDROSIS OF THE CAPITELLUM (PANNER'S DISEASE)*

REPORT OF A CASE

By E. W. KLEIN, LT MC USN†
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OSTEOCHONDROSIS of the capitellum humeri is a rare disease among aseptic epiphyseal necroses. Panner[6] reported the first 2 cases in 1929; Laurent and Lindstrom[3] reviewed the literature up to 1955 and recorded 21 cases. The author has been able to trace only a few publications dealing with this entity in the recent literature.

The majority of cases so far reported have been in children aged 4 to 10 years, who, with few exceptions, were males. The mean age was 8 years. There may be a history of relatively mild preceding trauma or there may be merely a gradual onset of discomfort in the elbow. Symptoms of mild local edema and tenderness on the medial aspect of the cubital articulation are pres-

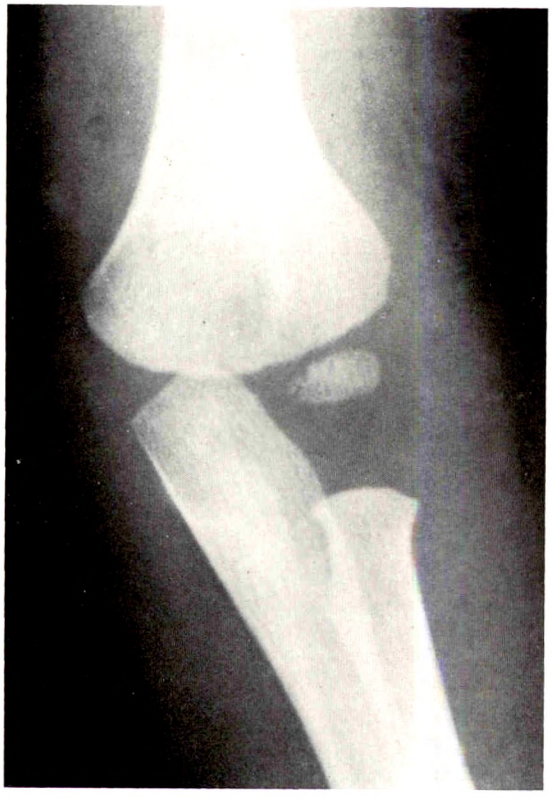


FIG. 1. *January 19, 1959:* Roentgenogram of the right elbow showing thinning of the cortex with signs of early fragmentation.

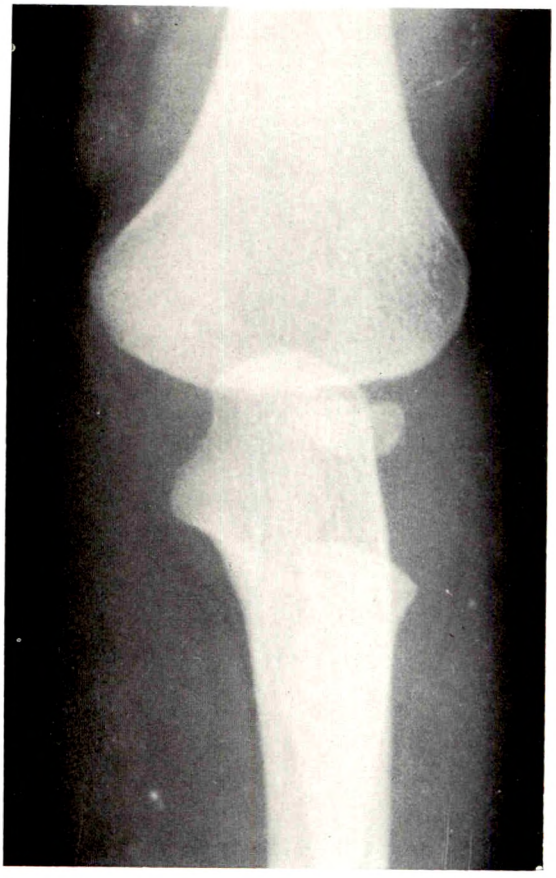


FIG. 2. *January 19, 1959:* Roentgenogram of the left elbow showing no changes.

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The opinions expressed are those of the author and do not necessarily represent the views of the Navy Department.
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ent. The roentgenograms reveal irregularity in the shape and structure of the capitellum with thinning of the cortex. A subcortical zone of rarefaction may be seen. Within the epiphysis are seen irregular zones of bony condensations, and at a later stage fragmentation and recalcifications as well as reduced thickness of the epiphysis are also noted. The changes thus correspond with those encountered in the Legg-Calvé-Perthes disease. As the process heals, which it does spontaneously and apparently irrespective of treatment,[1] a fullness of the anatomic part is restored. The clinical symptoms and signs disappear over a period of a few weeks; however, the roentgen manifestations are not commensurately improved and may persist for 1 to 3 years before complete restitution is seen. In later life there does not seem to be any residual distortion to mark the former existence of an avascular necrosis. Theoretically, however, such an elbow may be more susceptible in later years to the development of secondary hypertrophic or traumatic arthritis.[4]

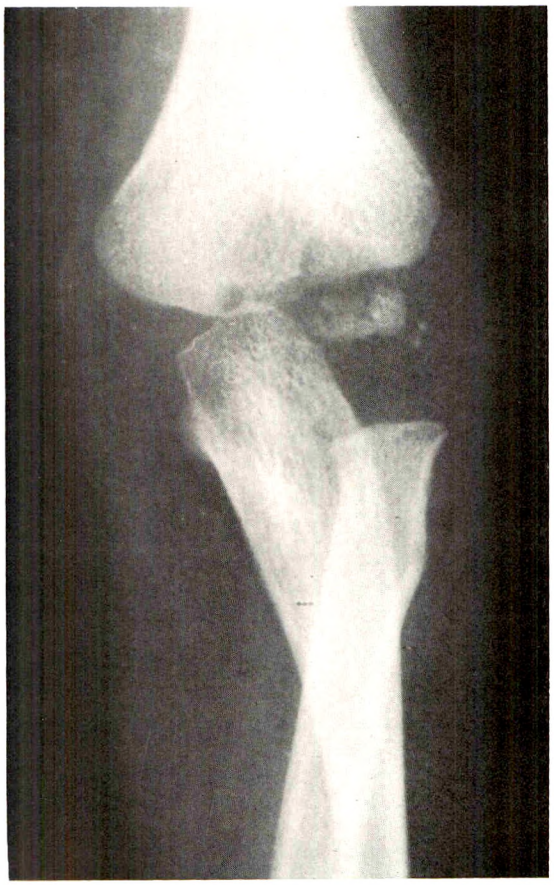


Fig. 3. *February 27, 1959:* There is marked irregularity in the shape and structure of the capitellum. Local edema is seen.

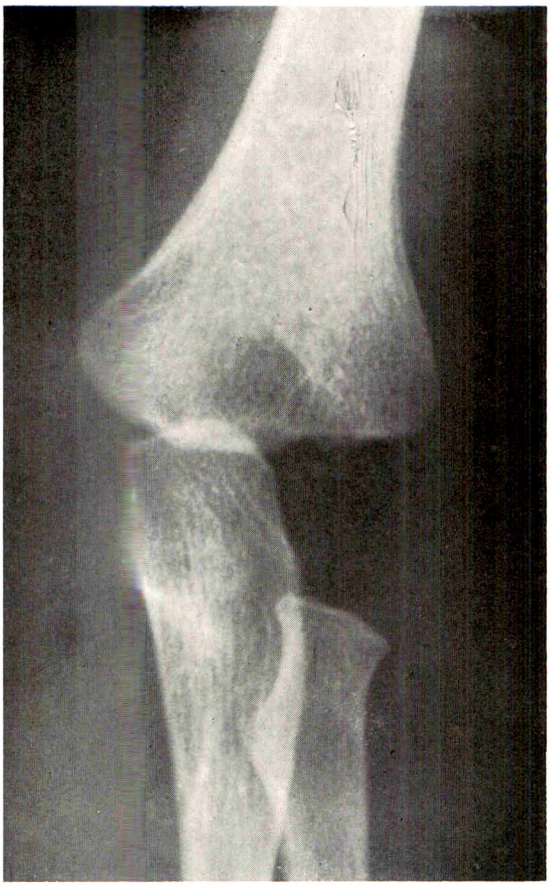


Fig. 4. *April 16, 1959:* Three months from onset the capitellum is essentially disintegrated.

REPORT OF A CASE

A 2 year old white girl was admitted to the hospital for evaluation of a right elbow in which swelling and pain had been present for 5 days. There was a progressive disability in usage. The past history was noninformative with the exception of a bout of tonsillitis 3 weeks prior to admission from which the child recovered satisfactorily.

Physical examination revealed a pale child weighing 24 pounds. The body temperature was 99.8°F. The ear, nose and throat examination and that of the heart and lungs were normal.

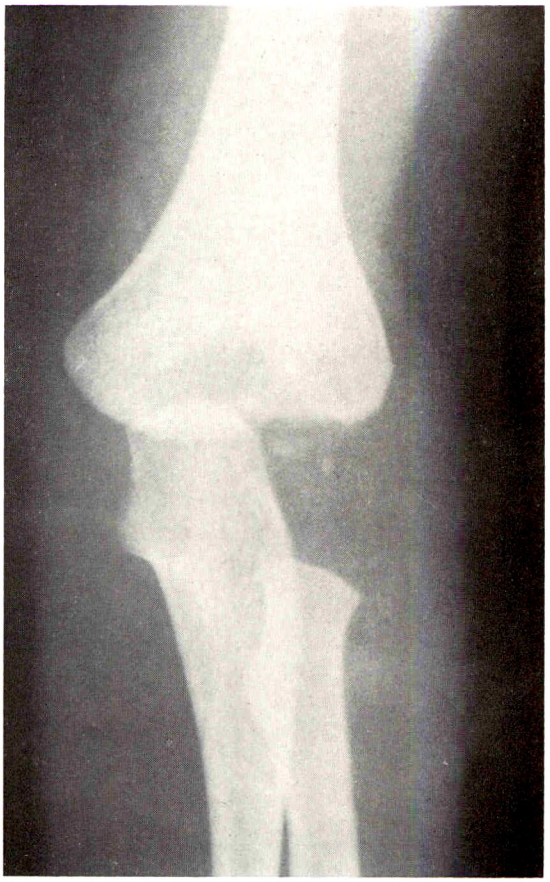


FIG. 5. *July 10, 1959:* Six months from onset there is evidence of spotty recalcification.

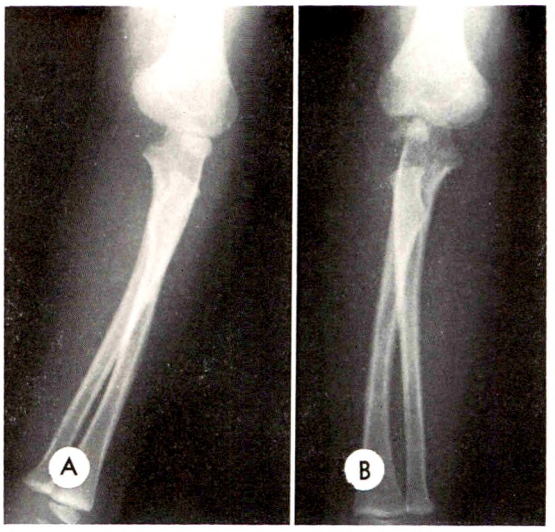


FIG. 6. *September 14, 1961:* (*A*) Left elbow. (*B*) Right elbow. Two and one-half years from onset, there is considerable recalcification which continues to be irregular in outline. The left elbow remains normal.

The right elbow was tender, swollen and warm to the touch, particularly over the olecranon process. The child was unable to fully extend the elbow. There was no other discernible joint involvement.

Indicated laboratory studies revealed a white blood cell count of 7,200; hemoglobin 12.6 gm., and a corrected sedimentation rate of 9. Roentgenograms of the elbow were interpreted as being normal. Repeated blood cultures were positive on 2 occasions for *Streptococcus pyogenes*. The skin tests were negative.

During the hospital stay the child continued to have an elevated temperature in spite of adequate antibiotic coverage. On the recom-

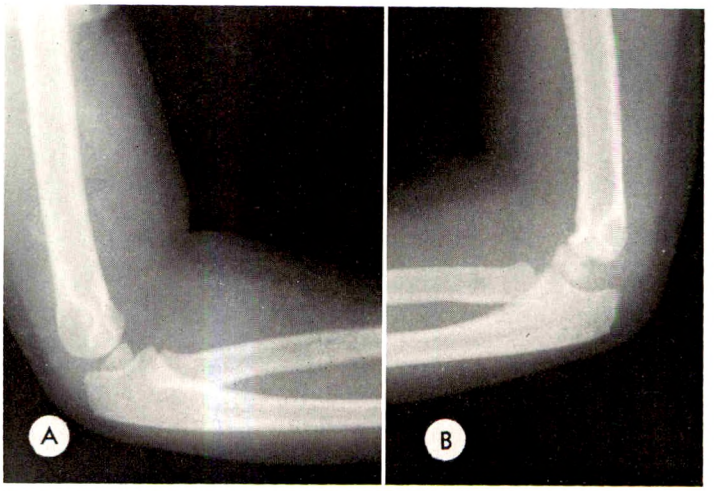


FIG. 7. *September 14, 1961:* (*A*) Left elbow. (*B*) Right elbow. Same as Figure 6, *A* and *B*. Lateral views.

mendation of the orthopedic consultant, the right elbow was prophylactically immobilized in a plaster cast. The patient was subsequently discharged when her temperature stabilized.

One month later the child was seen in the orthopedic clinic and roentgenograms of the elbow with the cast removed showed slight irregularity of the capitellum. There was no evidence of osteomyelitis. A monoarticular arthritis, cause unknown, was thought to be the most likely diagnosis.

The child continued to have mild symptoms in her right elbow and 2 months later symptoms developed in the left elbow. At this time roentgenograms of the right elbow showed very definite de-ossification and fragmentation of the capitellum consistent with an aseptic necrosis of this epiphysis. Figures 1–5 show the changes in the right capitellum over a 6 month period. No changes were noted in the left capitellum. The child was then presented to the orthopedic conference, and it was the consensus of opinion that this represented Panner's disease.

Re-examination $2\frac{1}{2}$ years later by roentgenograms (Fig. 6 and 7) showed regeneration of the capitellum. The cortex remained fragmented.

The child is doing well and is essentially asymptomatic.

SUMMARY

1. An unusual case of Panner's disease in a 2 year old girl is presented. A 2 year period of follow-up is covered by serial studies.

2. The clinical and roentgenographic aspects of the disease are discussed.

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CHONDROANGIOPATHIA CALCAREA SEU PUNCTATA*

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CHONDROANGIOPATHIA calcarea seu punctata is a rare malady of infancy that is classified with the chondrodystrophies. It is sharply differentiated from the others of this group by specific roentgen findings. These consist of multiple discrete punctate areas of calcification in the epiphyses. The disease was first described in 1914 by Conradi.[2] In 1952, Savignac[7] reported a case and in reviewing the literature could only find 22 other such cases. Several cases have since been recorded but the total number is still low. It has been estimated that the incidence is in the order of 1 in 1,500,000 births.[3] This syndrome has been described under a host of different terms. The more common ones are chondrodysplasia calcarea, chondrodystrophia calcificans congenita, punctate epiphyseal dysplasia and epiphyseal dysplasia puncticularis. Although the synonyms are many, there is no difficulty in recognizing the basic disorder. The disease is seen in newborn infants. Most of the cases reported have been in stillborns or in infants that have not survived the first year of life. There is no evidence of a sex linkage but a familial tendency has been pointed out.[3]

The most striking finding is that of micromelia. This may involve any of the extremities although rarely all in one infant. The proximal segment is involved most commonly in the reported cases. In the case to be presented, both the right arm and leg were shortened. This gave the startling impression such as seen in plastic toys when different size "halves" are fused together. Less striking but usually present are flexion deformities. Some of these are explained on the basis of muscle fibrosis. The larger joints such as the knee, elbow



FIG. 1. Roentgenogram of both forearms at the age of five weeks showing shortening of the right radius and punctate calcifications of the right wrist.

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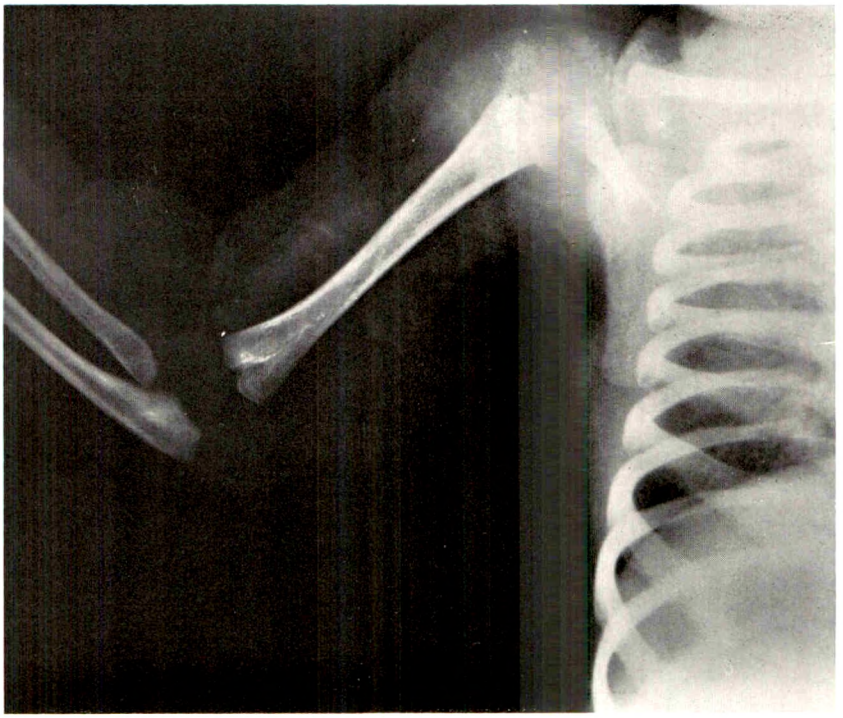


Fig. 2. Roentgenogram of the right humerus at the age of five weeks showing shortening with punctate calcifications in the proximal epiphysis.

and hips are those that are generally involved. At least these joints are more obvious in a stillborn. Other findings reported are less frequently seen and are of lesser diagnostic importance. Congenital bilateral cataracts have been noted but are not of unusual character. A dermatitis has been frequently described. It usually consists of a mild erythema with a scaley appearance that is not especially distinguishing. Still other findings that are described probably represent individual manifestations rather than a disease characteristic.

The roentgen findings are striking in appearance and the diagnosis is made on this basis. Typically, the epiphyses show multiple calcified areas which are stippled or punctate. These, of course, are the prime findings and lend themselves descriptively



Fig. 3. Roentgenogram of the lower extremities at the age of five weeks showing punctate calcification of the right distal femoral epiphysis and the right tarsal bones.



FIG. 4. Roentgenogram of the right upper extremity at the age of seven months showing continued shortness of the right radius with a tendency of the disorganized punctate calcifications of the right wrist to heal.

to the many synonyms given this condition. All cartilaginous bones can be affected. In addition, cartilage which ordinarily does not calcify, such as the thyroid and hyoid, and cartilaginous material, such as the intervertebral disks, which does not calcify until late in life may also be involved. In many cases, some of the features of classic chondrodystrophy are present. Why the systemic disturbance is incompatible with life in so many of these cases has not been ascertained.

Pathologically, there is a disturbance in vascularity of the epiphyseal cartilage and the cartilaginous anlage of the small bones of the wrist and ankle. These structures become the foci of the punctate calcareous deposits. The basic defect is as yet unknown. A maternal prenatal predisposing factor has not been established. Most of the cases described are in stillborns or in infants who did not live past the first year of life; however, there has been an increasing number of cases reported that have survived.[1,4,5,6,8] In these cases, the epiphyseal lesion disappears and there is often no residual finding. The case being presented here is of interest because of the rarity of this disease and also because the patient has survived infancy and is seven years old at the time of this writing.

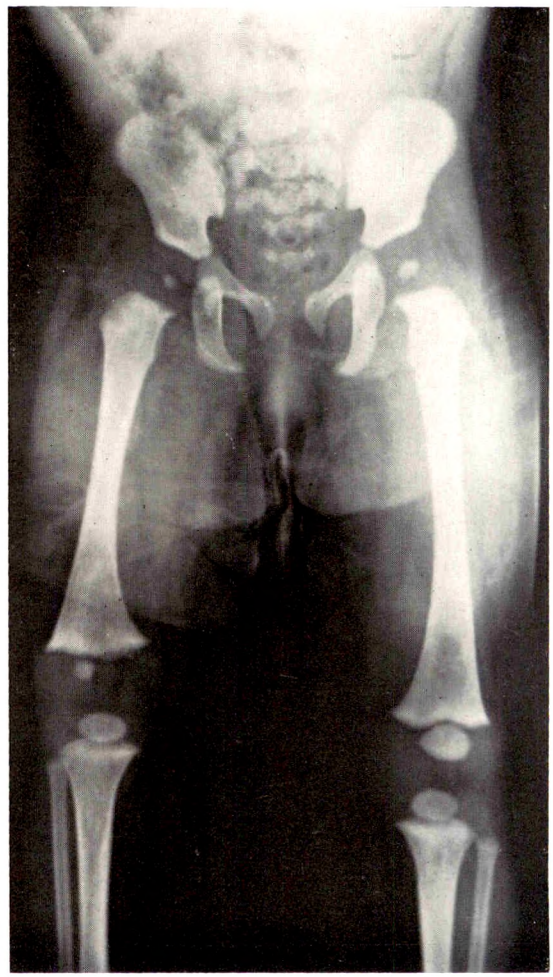


FIG. 5. Roentgenogram of the pelvis and femora at the age of seven months showing continued shortness of the right femur with some absorption of the dense calcific deposits.

REPORT OF A CASE

T.R., a Negro female infant, was admitted to the St. John Hospital on January 28, 1953, at the age of five weeks. She was delivered at a nearby hospital by a normal spontaneous delivery. Her birth weight was 9 lb., 4 oz. The hospital stay was entirely normal. The mother presented the following complaints: the child was extensively covered with large blue areas and had a swelling of the right arm and cried when it was handled. The family history was essentially negative. The mother was twenty-one years of age, the father was thirty-one. Both are living and well. There were 2 siblings aged seven and eight years, both living and well. There were no miscarriages or hereditary disease in the family. During the first trimester of this pregnancy, the mother vomited frequently and was confined to bed much of the time. The last trimesters were apparently normal.

The mother had a fourth child born on March 24, 1958, who is normal in all respects.

Physical Examination. The physical examination showed a well developed and well nourished infant in no distress. The weight was 10 lb., 14 oz. There was an abundance of mongolian spots which were very striking. The general examination was essentially normal other than the appearance of the limbs. There was a soft tissue enlargement of the upper anterior surface of the right forearm which extended about half the distance to the wrist. It was sharply demarcated by a deep crease, probably by a band of fibrous tissue. It was not hot, tender or indurated. It apparently was formed of skin and subcutaneous tissue. The right arm and right leg were obviously shorter than the left. This gave a peculiar appearance as if the right side of the body came out of a mold a few sizes smaller than the left. The right forearm measured $2\frac{3}{4}$ inches in length, the left measured $3\frac{1}{2}$ inches. The right lower extremity was 8 inches long and the left measured $9\frac{1}{2}$ inches. The right thigh measured $2\frac{1}{2}$ inches in length and the left thigh $3\frac{1}{4}$ inches. The right arm was externally

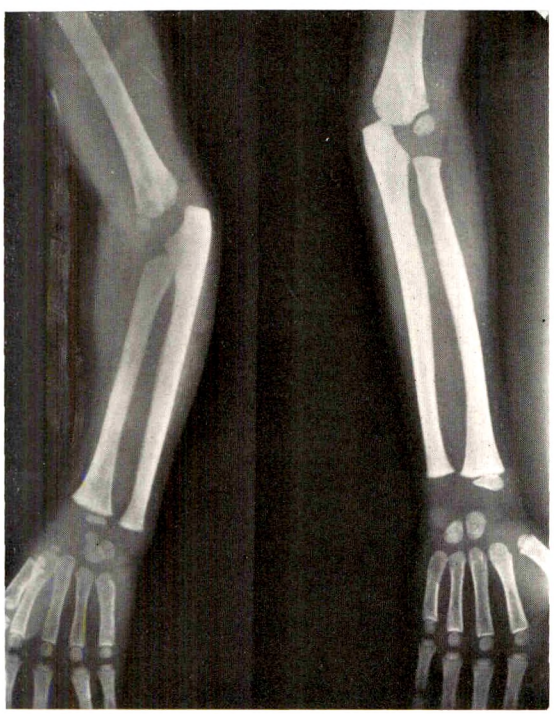


Fig. 6. Roentgenogram of both forearms and hands at the age of three years showing still some shortness of the right radius, but an almost normal appearance of the right wrist.

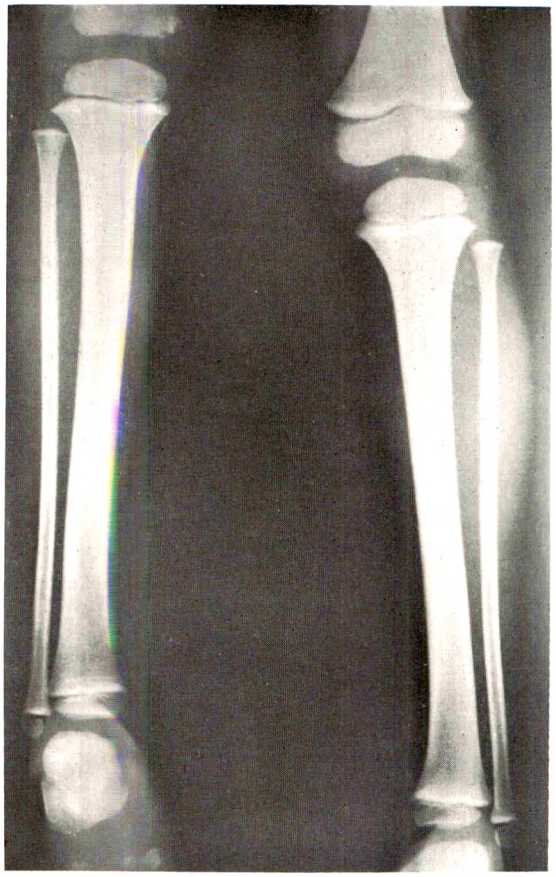


Fig. 7. Roentgenogram of both legs at the age of three years showing an almost normal appearance of the tarsal bones.

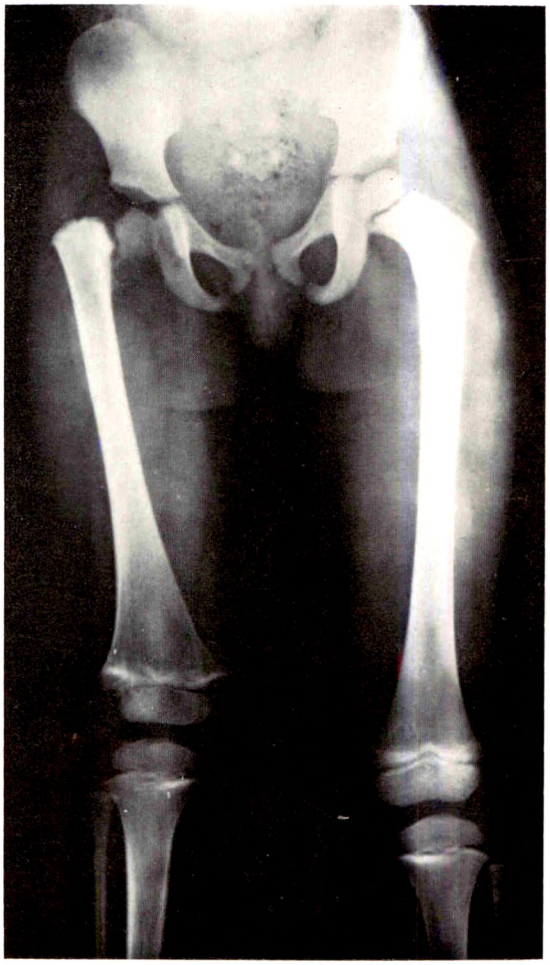


FIG. 8. Roentgenogram of the pelvis and femora at the age of three years showing a definite coxa vara of the right hip. The distal right femur is almost normal, except for irregularity of the metaphyseal end and a decrease in size of the epiphysis.

rotated with the fingers lying flaccid, although the child did move them on stimulation.

Laboratory Data. The following laboratory data were obtained: The Kahn test was negative. The sickle cell preparation showed no sickling after twenty-four hours. Serum calcium was 11.4 mg. per cent, serum phosphorus 7.3 mg. per cent (serum was hemolyzed), alkaline phosphatase 12.1 Bodansky units. The Sulkowich test was abnormally low on the first day but was reported as low to normal on the second specimen. Complete blood cell count showed the hemoglobin 13.0 gm., red blood cells 4.43 million, white blood cells 8,800, with polymorphonuclears 16 per cent, lymphocytes 83 per cent and monocytes 1 per cent. The protein bound iodine was reported 7.0 μg. The child's hospital course was uneventful. She was discharged home and has been followed closely for the past seven years.

Follow-up. At the age of four and one-half months, the patient erupted her two lower central incisor teeth. The others appeared during the average time range. Ophthalmic examination was entirely normal in the hospital and at a later age. At the age of two years, the dermatologist diagnosed a mild case of ichthyosis hystrix. Complete neurologic examination was normal. At the present time, the right arm is entirely normal other than a mild radial deviation. At the age of three and one-half years a diagnosis of coxa vara was made on the right side and a subtrochanteric osteotomy was performed. Up to this time, the right leg is still 1½ inches shorter than the left.

Roentgen Findings. The roentgen examination at five weeks (Fig. 1, 2 and 3) disclosed a right unilateral shortening of the radius, humerus, and femur. The shortening was most apparent in the humerus and femur. There was a disorganized punctate calcification of the right wrist, proximal humeral epiphysis, distal femoral epiphysis and the tarsal bones.

Progress roentgenograms made at seven months of age (Fig. 4 and 5) showed a tendency for the punctate epiphyses to heal in an organized fashion with some absorption of the dense calcified deposits. There was continued shortness of the right radius, humerus and femur. At this time a question of congenital coxa vara of the right hip was considered.

Roentgenograms taken at three years of age (Fig. 6, 7 and 8) showed an almost normal appearance of the right wrist and tarsal bones. A definite congenital coxa vara has developed at the right hip. The distal femoral area had now approached the normal with expansion of the metaphyseal area and irregularity of the metaphyseal space. The epiphysis was almost normal although smaller in size.

SUMMARY

A case of chondroangiopathia calcarea seu punctata is presented. This case shows the typical clinical and roentgen findings which are described in the literature and is an added example to the growing list of those who survive the disorder. The sequence of the disappearance of the pathologic lesions

is shown and the residual deformities are discussed.

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APPEARANCE AND FUSION OF OSSIFICATION CENTERS IN THE HUMAN SKELETON*

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WHEN the problem of a child's size, whether large or small, is brought to the physician's attention, an evaluation of the maturational status of the child's skeleton is part of the medical investigation. A knowledge of the wide variation in the level of ossification and fusion of the primary and secondary centers of the human skeleton which is compatible with health is essential in order to make an accurate evaluation of the child's growth pattern. Various methods have been offered by which an individual's skeletal age may be estimated. Different parts of the skeleton such as the hand, wrist, elbow, knee or hip have proved to be useful aides in this determination.

The purpose of this paper is to present the range in the ages at which ossification or fusion of the centers of these segments of the skeleton takes place as recorded from the study of the roentgenograms of the extremities of a group of healthy children.

REVIEW OF LITERATURE

There are in the literature numerous references citing ages at which the primary and secondary ossification centers of the human skeleton ossify and fuse as maturation progresses. More consideration has been given to the ages at which these centers ossify than to the ages at which fusion between epiphysis and diaphysis occurs. In most instances the ages given today for fusion are the same as those published in the original anatomy texts.[7] It was felt at that time that the shadows seen on roentgenograms often were confusing and unreliable and these early data were obtained from a direct study of cadavers and, therefore, were limited in number. Little attempt was made to separate the sexes. In 1931 Shelton[8] stated, "It is my belief, however, that no great difference occurs in the normal before the thirteenth birthday and that for practical purposes one table for both sexes, considering the variation allowed, is adequate."

On the chart published by Camp and Cilley[2] in 1931, and later revised by Girdany and Golden,[6] the sexes are separated for many of the ages of beginning ossification of the centers but a single age for the combined sexes is quoted for fusion of the secondary centers.

Probably the most complete set of data to be found is that published by Flecker[4] in 1942. This work is based on the appearance and fusion of primary and secondary centers as seen in roentgenograms. All the centers of the skeleton are tabulated by sex according to the age at which they begin to ossify or fuse. About 50 fetuses ($8\frac{1}{2}$ to 38 weeks) were studied for information concerning the primary centers which are ossified before birth. It might be pointed out that the lateral epicondyle, capitellum and trochlea are shown here to fuse with each other to form a conjoint epiphysis which then joins the shaft of the humerus. This point has been made repeatedly throughout the literature. According to Francis,[5] the lateral epicondyle is a separate center in about half of the cases and is continuous with the capitellum in the rest. The trochlea is always a separate center but in every case these three centers are reported to unite before fusion to the diaphysis of the humerus takes place. The epiphyses for the head of the humerus and the lateral head

* From the Child Research Council and the University of Colorado School of Medicine.
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or greater tuberosity also have been found to fuse with each other before uniting with the proximal end of the humerus.

MATERIAL

The longitudinal growth study of the Child Research Council includes the study of growth and maturation of the human skeleton by means of roentgenograms of certain parts of the skeleton. The left upper and lower extremities are roentgenographed at 2 months, 4 months, and 6 months and at 6 month intervals thereafter to maturity. The child is small enough during his first year of life so that the hand and foot are included on the early roentgenograms. Separate roentgenograms of the left hand are then taken every 6 months from 1 year to maturity. No further roentgenograms of the foot are taken.

A total of 207 children, 102 boys and 105 girls, comprise the group which is being or has been roentgenographed in this manner. Not all of these children have a complete series of roentgenograms. Since their ages range from 5 months to 35 years, some are too young to have had more than 2 sets of long bone roentgenograms and others were near adolescence before this part of the study was instituted.

The ages at which the carpal centers ossify and at which the epiphyseal centers that are seen on these particular roentgenograms ossify and later fuse to their diaphyses have been tabulated according to sex. Fusion was based on complete union between epiphysis and diaphysis, although the epiphyseal plate still might be visible (Sidhom and Derry,[9] Stage 2). These ages are found in Tables I and II. In Figure 1 the ages are presented in the form of bar diagrams. The bar in each case represents the span in years between the earliest age at which a particular center ossifies or fuses and the latest age at which these events take place. The upper bar indicates the range for girls and the lower bar the range for boys. The arrow with each bar marks the median age of ossification or fusion of the center in question. The centers are listed in order according to the median age from earliest to latest in which they are found to ossify in this group of girls. The number of children whose roentgenograms have been examined for the ossification of each center at each age varies from 44 to 62 for boys and from 45 to 75 for girls. When fusion of the epiphyses is considered, the numbers are somewhat smaller, with 19 to 63 for boys and 27 to 71 for girls. The figure at the beginning or end of each bar is the number of children in that group.

DISCUSSION

Since roentgenograms are not taken in the newborn period as a part of this study, the earliest age at which ossification can be recorded is 2 months. The first center to ossify in both sexes, and which is present on 100 per cent of the roentgenograms made at 2 months, is the epiphysis for the distal end of the femur. The epiphysis for the proximal end of the tibia is the second center to ossify in this series and is visible in 100 per cent of the girls at 2 months and 98 per cent of the boys of this age. The tarsal cuboid is third and is seen at 2 months in 98 per cent of girls and 91 per cent of boys. The fourth center to appear in this series is the epiphysis for the head of the humerus and it is present in 81 per cent of 2 month old girls and 78 per cent of 2 month old boys.

As maturation progresses, the boys lag slightly behind the girls until about the time the medial epicondyle of the humerus ossifies in girls, after which there is an increasing age difference in ossification time between sexes, as seen in Figure 1. For the remaining centers diagrammed, a difference of 2 to 3 years is the most common in this group. The two sexes maintain approximately the same order of ossification until the medial epicondyle appears in girls. In general, in this group of children, fusion of the secondary centers takes place 2 to $2\frac{1}{2}$ years later in boys than in girls. The order in which centers fuse varies very little between sexes.

When the epiphyses for the distal end of

TABLE I

MEDIAN AGES IN YEARS AND MONTHS AND RANGES IN AGE AT WHICH CERTAIN OSSIFICATION CENTERS APPEAR IN A GROUP OF HEALTHY CHILDREN

Center		Median	Girls' Range	No. of Cases	Median	Boys' Range	No. os Cases
Capitate		0–2.5	0 to 1–0	57	0–3	0 to 1–0	49
Hamate		0–3	0 to 1–0	57	0–5	0 to 2–0	49
Triquetrum		2–1	0–2 to 5–0	62	3–1	0–4 to 6–0	56
Lunate		3–1	0–6 to 6–6	62	4–7.5	0–4 to 8–0	57
Greater multangular		4–2	2–0 to 7–6	57	6–4	3–6 to 10–0	54
Lesser multangular		4–6	1–6 to 7–0	59	6–10	4–0 to 10–6	62
Navicular		4–6	2–0 to 7–6	57	6–10	3–0 to 9–6	54
Pisiform		8–7.5	5–0 to 11–0	64	11–8	8–6 to 14–6	55
First metacarpal sesamoid		11–3	8–6 to 14–0	55	13–8	11–0 to 16–0	51
Tarsal cuboid		98% by 0–2	0 to 0–4	45	91% by 0–2	0 to 0–6	44
Metacarpal	I	1–11	0–6 to 3–0	72	3–1	1–6 to 6–0	51
	II	1–4	0–6 to 2–0	71	1–10	0–6 to 3–0	53
	III	1–4.5	0–6 to 2–6	74	2–0	0–6 to 3–6	53
	IV	1–6	0–6 to 2–6	74	2–1.5	1–0 to 4–0	54
	V	1–7	0–6 to 3–0	73	2–5	1–0 to 4–0	54
Proximal phalanx	I	2–1.5	1–0 to 3–0	69	3–4.5	1–0 to 5–6	49
	II	1–2	0–4 to 2–0	70	1–7.5	0–6 to 2–6	55
	III	1–1.5	0–4 to 2–0	72	1–7	0–6 to 3–0	55
	IV	1–2	0–4 to 2–0	71	1–8	0–6 to 3–0	54
	V	1–5.5	0–6 to 2–6	73	2–1.5	1–0 to 4–0	54
Middle phalanx	II	1–8	0–6 to 3–0	64	2–6	1–0 to 4–0	49
	III	1–7.5	0–6 to 2–6	69	2–4	0–6 to 4–0	49
	IV	1–7.5	0–6 to 2–6	69	2–5	1–0 to 4–0	52
	V	2–3	0–6 to 4–0	65	3–7	1–6 to 5–6	50
Distal phalanx	I	1–5	0–4 to 2–6	63	1–10.5	0–6 to 4–0	50
	II	2–5	0–6 to 3–0	58	3–7	1–0 to 6–0	45
	III	1–11	0–6 to 3–0	65	2–7	1–0 to 4–0	48
	IV	2–0	0–6 to 4–0	66	2–8	1–0 to 4–0	48
	V	2–4	0–6 to 3–6	63	3–7	1–6 to 5–0	48
Humerus—head		81% by 0–2	0 to 0–4	58	78% by 0–2	0 to 0–6	45
Greater tuberosity		0–9	0–2 to 2–6	68	1–2	0–4 to 4–6	53
Capitellum		0–7	0 to 1–6	69	0–10	0 to 2–6	56
Lateral epicondyle		9–10	7–6 to 12–0	70	12–5	9–6 to 15–6	61
Trochlea		9–5	5–6 to 12–6	70	10–7	7–0 to 14–0	58
Medial epicondyle		3–10	2–0 to 6–6	64	7–1	4–6 to 10–0	60
Radius—head		4–10	2–0 to 8–0	70	6–3	2–6 to 9–6	57
Olecranon		8–8	6–0 to 11–6	69	11–3	7–6 to 14–0	54
Distal radius		1–1.5	0–4 to 3–0	69	1–4	0–4 to 3–6	53
Distal ulna		6–0	3–6 to 9–0	75	7–5	5–0 to 10–0	62
Femur—head		0–5	0 to 1–0	67	0–6	0–2 to 1–0	55
Greater trochanter		2–10	1–6 to 4–0	68	4–0	2–0 to 6–0	60
Distal femur		100% by 0–2	0 to 0–2	58	100% by 0–2	0 to 0–2	47
Proximal tibia		100% by 0–2	0 to 0–2	58	98% by 0–2	0 to 0–4	47
Proximal fibula		3–1.5	1–0 to 6–6	60	4–5	2–0 to 6–6	57
Distal tibia		0–5	0 to 1–0	68	0–5.5	0–2 to 1–0	56
Distal fibula		1–0.5	0–4 to 3–0	69	1–4	0–4 to 2–6	50

TABLE II

MEDIAN AGES IN YEARS AND MONTHS AND RANGES IN AGE AT WHICH CERTAIN EPIPHYSEAL OSSIFICATION CENTERS FUSE WITH THEIR DIAPHYSES IN A GROUP OF HEALTHY CHILDREN

Center		Median	Girls' Range	No. of Cases	Median	Boys' Range	No. of Cases
Metacarpal	I	14–1	11–6 to 16–0	31	16–4	14–0 to 18–6	32
	II	14–6	11–6 to 17–0	31	16–6	14–6 to 18–6	31
	III	14–6	11–6 to 17–0	31	16–6	14–6 to 18–6	31
	IV	14–5	11–6 to 17–0	31	16–5	14–6 to 19–6	31
	V	14–5	11–6 to 16–6	31	16–6	14–6 to 19–6	31
Proximal phalanx	I	14–2	11–0 to 17–0	33	16–3	14–6 to 18–6	33
	II	14–2	11–0 to 16–6	31	16–4.5	14–6 to 18–6	32
	III	14–2	11–0 to 16–6	32	16–4	14–0 to 18–6	32
	IV	14–3	11–0 to 16–6	33	16–6	14–6 to 18–6	32
	V	14–2	11–0 to 16–0	32	16–3	14–6 to 18–6	33
Middle phalanx	II	14–4.5	11–0 to 17–0	33	16–5	14–6 to 19–6	30
	III	14–4.5	11–0 to 17–0	31	16–6	14–6 to 19–6	31
	IV	14–4	11–0 to 17–0	31	16–5	14–6 to 18–6	31
	V	14–3	11–0 to 17–0	33	16–4	14–6 to 19–6	30
Distal phalanx	I	13–8	10–6 to 15–6	36	15–11	13–6 to 18–0	35
	II	13–7	10–6 to 16–0	37	15–10	13–6 to 19–6	35
	III	13–7	10–6 to 16–0	37	16–0	13–6 to 18–0	34
	IV	13–8	10–6 to 16–0	35	15–10.5	13–6 to 18–0	34
	V	13–7	10–6 to 15–6	37	15–11	13–6 to 18–0	33
Humerus—head		15–7	13–0 to 17–0	27	18–2	16–6 to 20–0	19
Greater tuberosity		4–1	2–0 to 7–6	71	5–6	3–0 to 8–6	63
Capitellum		12–5	9–6 to 14–0	62	15–2	13–6 to 17–6	58
Lateral epicondyle		12–8	9–6 to 15–0	57	15–4	13–6 to 18–0	56
Trochlea		12–4	9–6 to 14–0	58	15–1.5	13–0 to 18–0	58
Medial epicondyle		14–1	11–0 to 16–0	51	16–4	14–0 to 19–0	45
Radius—head		13–6	10–6 to 16–0	57	16–2	14–0 to 19–0	47
Olecranon		12–8	10–0 to 14–6	58	15–4.5	13–6 to 18–0	56
Distal radius		15–10.5	13–0 to 17–0	28	18–0	16–0 to 20–0	21
Distal ulna		15–11	12–6 to 17–0	30	17–10.5	16–0 to 20–0	20
Femur—head		14–2	11–0 to 16–6	52	16–3	14–0 to 19–0	49
Greater trochanter		13–11	11–6 to 16–0	50	15–11	14–0 to 19–0	47
Distal femur		14–9	12–0 to 17–0	46	16–7.5	14–0 to 19–0	47
Proximal tibia		14–10	12–0 to 17–0	44	16–11	14–6 to 19–6	42
Proximal fibula		15–2	12–0 to 17–0	38	17–2	15–0 to 20–0	37
Distal tibia		14–10	12–0 to 17–0	44	16–10.5	14–0 to 20–0	46
Distal fibula		14–10.5	12–0 to 17–0	42	16–10.5	15–0 to 20–0	45

the humerus are examined for the children in this study, a great deal of variation in the manner in which the capitellum, trochlea and lateral epicondyle fuse is found. The often repeated statement that these centers fuse to form a conjoint epiphysis and then fuse as such to the shaft of the humerus rarely holds true. The three centers were found to unite with the humerus in several different patterns, including fusion as a single unit or as two parts or three separate centers.

The roentgenograms of the elbows of 59 girls and 56 boys were reviewed. Union or nonunion between the three centers was recorded according to the roentgenogram taken 6 months prior to the one showing the first evidence of fusion to the humeral

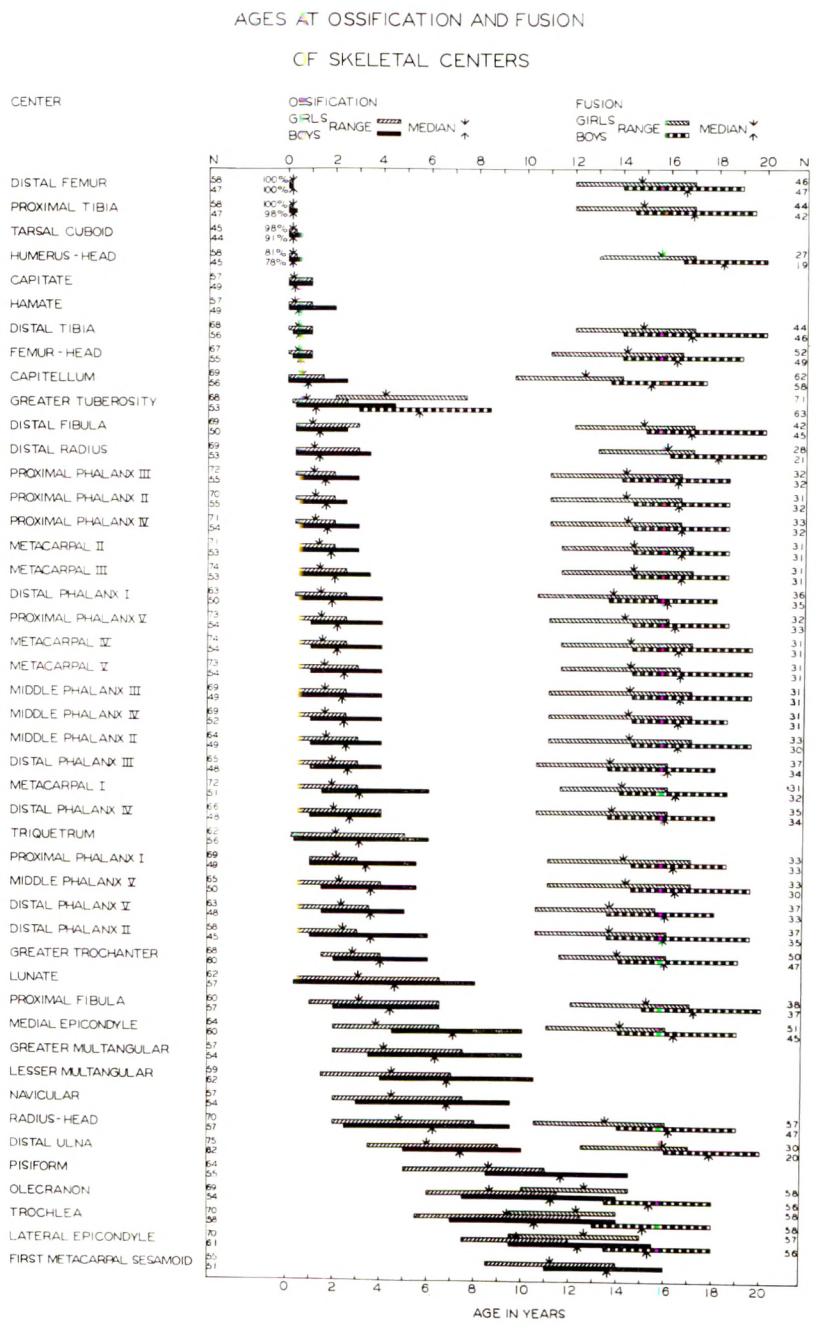


FIG. 1. Bar diagrams representing the ranges in age over which certain skeletal ossification centers appeared and fused in children of the Child Research Council study. The upper bar indicates the range for girls and the lower bar the range for boys. The median age of ossification or fusion of each center is indicated by the arrow. The centers are listed in order according to the median age from earliest to latest in which they are found to ossify in the girls of the group. The figure at the beginning or end of each bar is the number of children in that group.

shaft. It had been observed that, for 12 per cent of the girls in the group, the lateral epicondyle ossified as a continuation of the capitellum but in 88 per cent it appeared as a separate center. In 38 per cent of the boys this center originated in combination with the capitellum and in 62 per cent as a separate center.

In 11, or 19 per cent of the 59 girls and 25, or 45 per cent of the 56 boys, the capitellum trochlea and lateral epicondyle united to form a single center. Although the three centers in question united to one another prior to final fusion with the distal humerus, it did not necessarily mean that the final fusion occurred at the same age for the three component parts. In only 4 of these 11 girls and 9 of the 25 boys did this resulting center fuse to the shaft of the humerus as a unit, while in 1 of the 11 girls and 9 of the 25 boys each section of the conjoint epiphysis fused with the humeral shaft at an independent age. In 6 of the 11 girls and 7 of the 25 boys two of the three parts of the combined epiphysis (capitellum and trochlea or capitellum and lateral epicondyle) united with the humerus at the same age while the third part (trochlea or lateral epicondyle) united at a different age. Fusion between capitellum and lateral epicondyle was much more common in both sexes than fusion between capitellum and trochlea. The former occurred in 58 per cent of girls (34 out of 59) and 45 per cent of boys (25 out of 56). Although the two centers had united, in only 11 of the 34 girls and 8 of the 25 boys did they join the humerus as a whole; and in 23 of the 34 girls and 17 of the 25 boys the lateral portion fused with the shaft at a different and usually later age than did the part representing the capitellum. In 24 per cent, or 14 of the 59 girls and 11 per cent, or 6 of the 56 boys the lateral epicondyle remained a separate center. In contrast the trochlea remained a separate center in 78 per cent, or 46 of the 59 girls and 55 per cent, or 31 of the 56 boys, and fused with the capitellum in only 2 of the 59 girls and no boys. The capitellum and trochlea of one of these girls united with the distal humerus as a unit, but in the other girl fusion took place at two different ages. The capitellum remained a separate center throughout in 20 per cent, or 12 of the 59 girls and 11 per cent, or 6 of the 56 boys.

The epiphysis for the proximal end of the humerus ossifies from 2 centers, the head and lateral head or greater tuberosity. These 2 centers fuse during childhood to unite with the shaft of the humerus toward the end of adolescence.

Since most of the ages presented by earlier authors are not separated according to sex, it is difficult to make an exact comparison between these ages and the Child Research Council data. In general, however, there seems to be fairly good agreement between previously published ages of ossification[7] and the present findings. Vogt and Vickers,[12] Flecker[4] and Elgenmark[3] reported ages for ossification according to sex. These ages compare quite well with the Child Research Council median ages with significant differences being apparent in only a few instances. For several centers the ranges of ossification reported by the authors mentioned are narrower than the Child Research Council ranges.

The ages reported in the literature for the time of fusion of the epiphyses with their diaphyses are often later than those from the Child Research Council group. Todd[11] published, according to sex, the ages of fusion for the secondary centers of the extremities. For girls, about half of these ages agree quite well with the median ages in the present study. For boys, about two-thirds of the ages are in agreement.

Todd[11] also listed these epiphyses in the order in which they fuse to the long bones. The epiphyses for the head of the radius and the distal femur are the only centers which are "out of order" as compared to the present study. Other differences in order are insignificant.

Another series which reports ages of fusion for centers of the upper extremity in boys was published in 1929 by Borovanský

and Hněvkovský.[1] Here again the ages are in good agreement with the Child Research Council data.

Comparing Child Research Council median ages with Flecker's[4] group "e," *i.e.*, "ages at which in at least half of the individuals epiphyses were found to be fused," there is agreement within ±6 months in about one-third of centers in girls and in about one-fourth of centers in boys. The span of years over which the various secondary centers fuse is wider for the majority of centers in the present study than it is in the data reported by Flecker.

SUMMARY

Roentgenograms of the left extremity of healthy children are included in the longitudinal growth study of the Child Research Council. From these roentgenograms the ages at which the visible primary and secondary centers of the skeleton ossify and fuse to the long bones have been recorded. The age ranges over which the centers appear and fuse to their respective shafts are tabulated by sex as well as the median ages for ossification and fusion of these centers. One hundred two boys and 105 girls made up the group of children in this report. No newborn roentgenograms were taken, but the ossification center in the epiphysis for the distal end of the femur was present in 100 per cent of children aged 2 months. Also on the 2 month roentgenograms the ossification center in the epiphysis for the proximal end of the tibia was present in 100 per cent of girls and 98 per cent of boys and in the tarsal cuboid in 98 per cent of girls and 91 per cent of boys. Eighty-one per cent of 2 month old girls and 78 per cent of 2 month old boys had an ossified center for the head of the humerus. The ages at which ossification centers appeared in boys became progressively older as compared to girls until adolescence, when the last centers to ossify and the first centers to fuse did so 2 to 3 years later in boys than in girls. The order in which centers ossify and fuse varied little between sexes. Fusion of the three centers, capitellum, trochlea and lateral epicondyle, at the distal end of the humerus took place as 3 separate centers, as 2 centers, or as a single unit in this group. The epiphyses for the head of the humerus fused in all cases before uniting to the shaft.

Comparison of Child Research Council data with those in the literature shows fairly close agreement between ages of ossification but less agreement between ages of fusion of epiphyses to diaphyses.

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SCLEROSING OSTEOGENIC SARCOMATOSIS*

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OSTEOGENIC sarcoma involving primarily one osseous structure with progressive involvement of other portions of the skeleton is not in itself particularly unusual. However, the occurrence of what apparently is the development of multiple osteogenic sarcomata arising independently in multiple areas of the skeleton is extremely rare and the roentgenographic features of the sclerosing form of this condition are striking in their appearance. Recently we have had occasion to see 2 children with this condition and a review of the radiologic literature was rewarding in that we discovered a similar case reported by Moseley and Bass,[5] who also described 3 other similar cases occurring in children and reported by different authors.[1,3,4]

Because the roentgenographic features are essentially pathognomonic and because of the peculiar biologic behavior suggested by the multicentric origin of this type of neoplasm, we believe that presentation of the following case reports is justified. The distribution of the skeletal lesions in each case is shown in Figure 1, *A* and *B*.

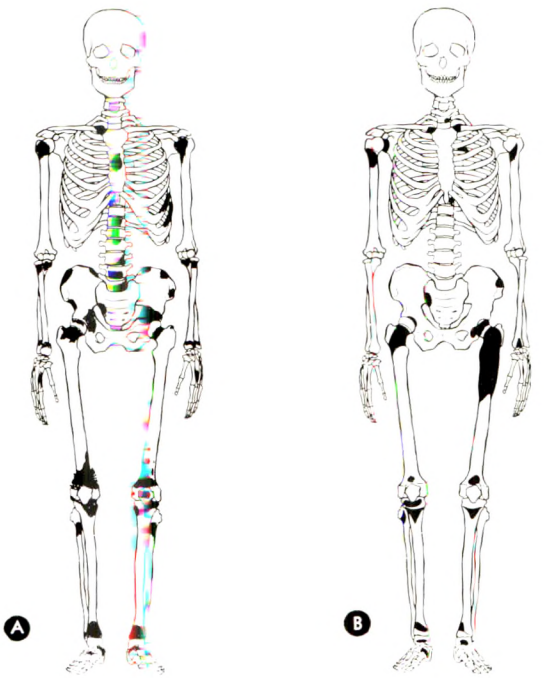


FIG. 1. (*A*) Case I: Distribution of lesions approximately eight weeks after onset of symptoms. (*B*) Case II: Distribution of lesions twelve weeks after initial complaint.

REPORT OF CASES

CASE I. D. J. H. was a six year old white girl who was well until July, 1959, when she developed pain in her right knee and, a few days later, in her right wrist. Roentgen examination of the right knee performed by her local physician showed "increased density" involving the osseous structure of the knee. This was misinterpreted as possibly being the result of "scurvy" and the patient was put on large doses of vitamin C without benefit. She was then referred to Texas Children's Hospital for further evaluation.

There was no history of trauma or heavy metal intoxication, although this was suspected because she had a "habit of putting objects in her mouth." The patient had not been on any type of medication other than vitamin C. The family's water supply was obtained from a new cistern composed of zinc with tar surfacing. There was no history of exposure to radiation or radioactive substances.

The past history was noncontributory. The patient had been in perfect health until the present illness.

The father had arrested pulmonary tuberculosis but the patient's mother had been in good health. There was no history of similar or related bone abnormalities.

Physical Examination. At the time of first admission the patient was alert and cooperative without evidence of acute or chronic illness. There was mild tenderness over the right knee

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joint but no evidence of swelling or redness. There were no other abnormal physical findings.

Laboratory Findings. At the time of admission the hemoglobin was 12 gm. per cent, hematocrit 35 per cent, white blood cell count 6,300 with 62 per cent segmental neutrophils, 7 per cent band forms, 23 per cent lymphocytes, 4 per cent monocytes and 4 per cent eosinophils. Peripheral blood smears showed slight polychromasia but no evidence of basophilic stippling of the red blood cells. The urinalysis and VDRL were negative. Blood chemistry studies showed urea nitrogen of 17 mg. per cent, CO_2 of 24.5 mm. per liter, potassium 4.1 mEq. per liter, chloride 105 mEq. per liter, creatinine 0.76 mg. per cent. The corrected sedimentation rate was 15 mm. per one hour. Total serum proteins were 7.3 gm. per cent with albumin 4.2 and globulin 3.1 gm. per cent. Calcium was 8.9 mg. per cent, phosphorus 5.4 mg. per cent and the alkaline phosphatase was greater than 50 Bodansky units. Febrile agglutinins and heterophile titer were negative. The SGOT was 37 and the SGPT 9. The urinary porphyrins were normal and a twenty-four hour urine sample examined spectrographically for heavy metals including lead and mercury was negative. Serum bilirubin was 0.3 mg. per cent total with 0.2 mg. per cent direct. Cephalin flocculation was negative at forty-eight hours and the thymol turbidity was 1.8 units. Skin tests for tuberculosis, histoplasmosis, coccidioidomycosis, and blastomycosis were all negative. There was no evidence of radioactivity as determined by a radioactivity detector.

Roentgen Findings. Roentgenograms of the skull and lungs were normal. Examination of the spinal column showed ill-defined areas of increased density involving several of the dorsal and lumbar vertebrae, but the intervertebral disk spaces appeared normal (Fig. 2*A*).

Examination of the pelvis showed multiple opaque areas involving the inferior portion of the sacrum at the right sacroiliac joint, as well as similar densities scattered along the crest of each ilium and above each acetabulum. Similar involvement of each proximal femoral metaphysis was identified (Fig. 2*B*).

Examination of the hands showed mild sclerosis of the right first metacarpal and the left navicular. Roentgenograms of the wrists revealed a homogeneous increase in density of the distal end of each radius and ulna. The epiphyses were of normal appearance and no abnormality of the epiphyseal line was identified. There was early periosteal new bone formation on the lateral side of the distal ulnar metaphysis (Fig. 2*C*). Examination of the elbows showed similar homogeneous density involving the left olecranon, the right elbow appearing normal. Roentgenograms of the shoulder girdles showed homogeneous density involving the proximal humeral metaphyses but without involvement of the epiphyses or scapulae.

The distal ends of the femora showed marked osteosclerosis (Fig. 2*D*). The density at the distal end of the left femur was more marked and extended cephalad into the distal end of the femoral diaphysis. On the medial aspect of this metaphysis there was extension of the sclerotic process into the subperiosteal region. A small, similar density 1 cm. in area was identified in the medial portion of the distal right femoral epiphysis. Bands of osteosclerosis were also present at the proximal end of the metaphysis of each tibia and fibula.

Examination of the ankles showed dense osteosclerosis paralleling the epiphyseal line but involving only the metaphyseal areas of the tibia and fibula (Fig. 2*E*). Discrete areas of increased density were also present in the right calcaneus, talus and scaphoid.

Histopathology. Five days after hospital admission a biopsy of the distal right femoral metaphysis was obtained consisting of several irregular fragments of bone measuring from 0.6 to 1.8 cm. in maximum dimensions.

Microscopically, there was a lack of uniformity in the architecture. The variation was dependent upon the presence or absence of irregular columns of osteoid tissue within broad sheets of pleomorphic cellular tissue (Fig. 3). In those areas where osteoid tissue was identified, there was a relatively sparse cellularity. Where the osteoid tissue was absent, cellularity was dense.

The individual cell forms varied from discrete round structures to those which were angulated or spindle-shaped. The cytoplasm was generally abundant. In most of the cells, nuclei were single, although occasional binucleated and multinucleated forms could be identified. The chromatin pattern was coarsely granular. Nucleoli were prominent but mitoses were very rare. A few telangiectatic vessels were present within both the cellular and acellular areas. Some of the spicules of osteoid tissue were calcified.

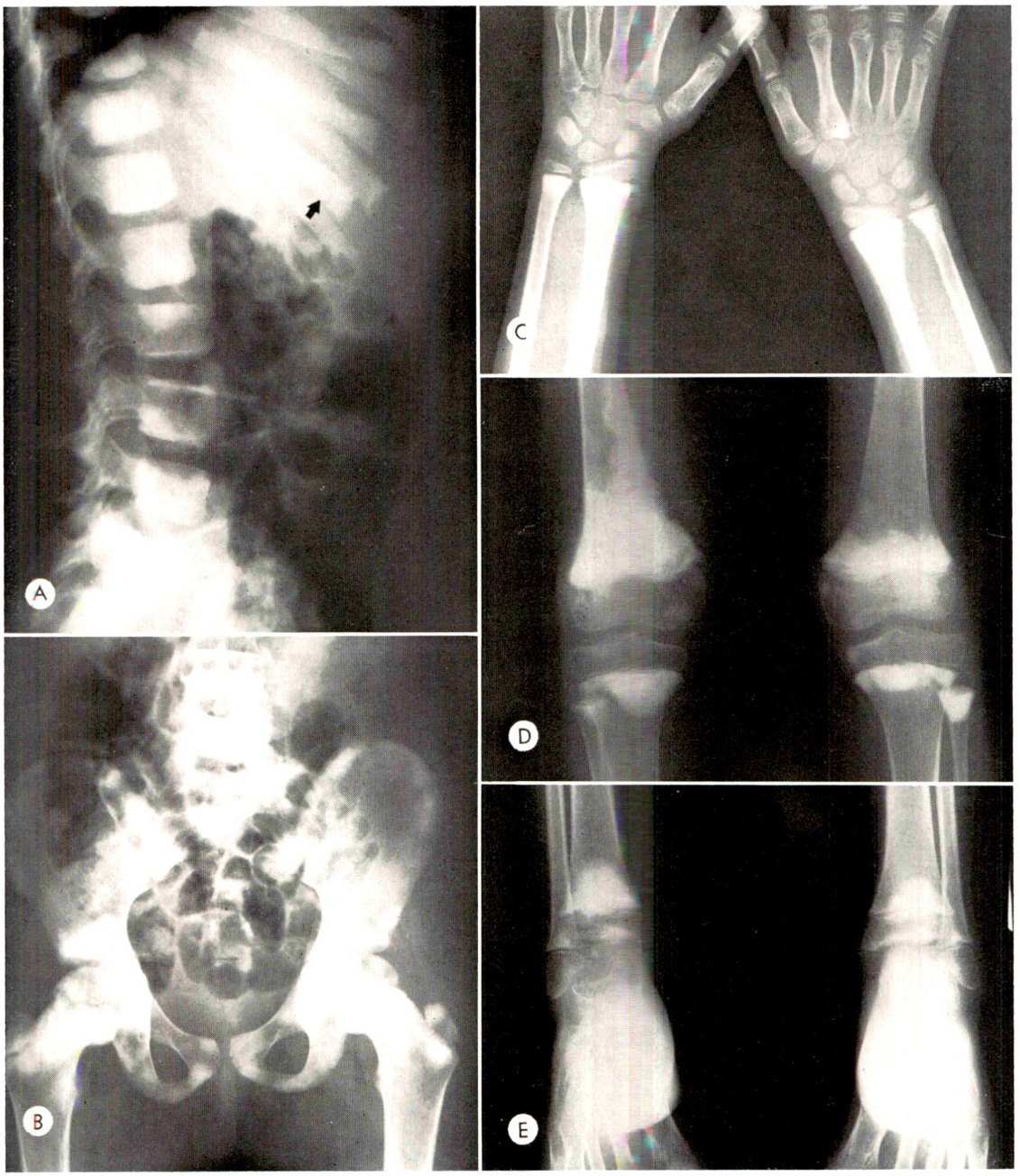


Fig. 2. Case 1. (*A*) Lateral roentgenogram of the lumbar spine shows multiple sclerotic vertebrae, as well as sclerosis and expansion of the anterior portion of one of the ribs (arrow). (*B*) Roentgenogram of the pelvis shows discrete areas of bone sclerosis, as well as involvement of the proximal portion of each femur. (*C*) Roentgenogram of the wrists shows osteosclerosis involving the distal radius and ulna bilaterally, as well as new bone formation affecting the distal radial and ulnar metaphyses on the right. (*D*) Roentgenogram of the knees shows similar areas of bone sclerosis affecting primarily the metaphyseal portions of the long bones. There is periosteal new bone formation involving the medial aspects of the distal right femoral metaphysis. (*E*) Roentgenogram of the ankles shows dense osteosclerosis paralleling the epiphyseal line but involving only the metaphyseal area of the tibia and fibula.

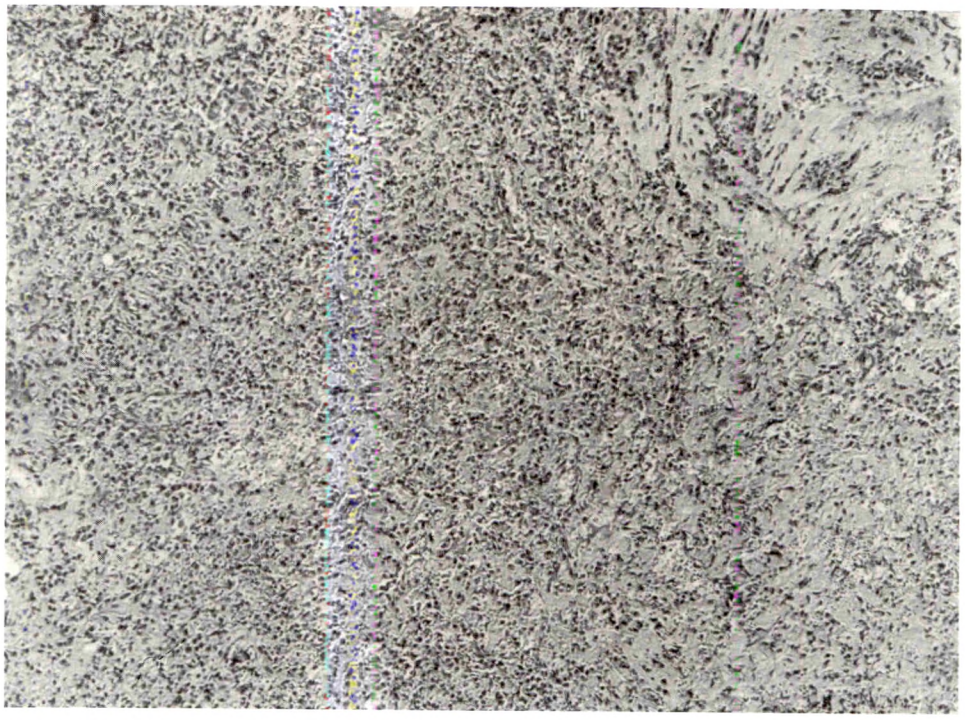


FIG. 3. Case 1. Photomicrograph of a biopsy specimen removed from the right femur shows broad sheets of pleomorphic elements irregularly interspersed with fragments of osteoid tissue. In other areas the proportions are reversed with dense accumulations of osteoid tissue and only few cells (H and E, 35×).

Within the overlying periosteum, the only evidence of reaction was the presence of a few hemosiderin-laden macrophages.

Three months after the initial biopsy, a bone marrow examination taken from the posterior iliac crest revealed a hypocellular marrow with a marked depletion of hematopoietic elements. There were numerous large mononuclear forms with an abundant pale foamy basophilic cytoplasm and a coarsely granular nucleus. Clear basophilic nucleoli were prominent and numbered from one to four within each cell.

Urinary sediment at that time contained many large mononuclear cells resembling those seen in the bone marrow. In many of these the chromatin pattern of the nuclei was finely granular, and nucleoli were abundant.

Clinical Course. Repeat roentgenograms of the skeleton made two weeks later showed considerable increase in the extent of all lesions. The patient was placed on cytoxan therapy under the direction of Dr. Donald Fernbach. She experienced noticeable relief of the discomfort involving the right knee, but mild side effects consisting of anorexia, nausea, and vomiting were noted. Alopecia occurred, as was expected, and the patient suffered a rather marked leukopenia which disappeared following stoppage of the drug. The alkaline phosphatase dropped to 18 Bodansky units. She was discharged on what was considered a maintenance dose of cytoxan consisting of 100 mg./day.

Two months later she was readmitted to the hospital because of low back pain and dysuria. Physical examination showed the child to have extensive pain in the lumbosacral area. Laboratory studies showed a hemoglobin of 9.1 gm. per cent, a white blood cell count of 3,000, reticulocytes 0.5 per cent, platelets 115,000, blood urea nitrogen 10 mg. per cent, and an alkaline phosphatase greater than 50 Bodansky units. Urinalysis showed the presence of many red blood cells as well as tumor cells. Roentgen examination of the skeleton showed an increase in the number and size of the osteoblastic lesions as well as pathologic fractures of D-12 and L-5. She was fitted with a Taylor back brace, which provided considerable symptomatic relief, and was continued on cytoxan therapy. She returned to her home town and succumbed from her malignancy one month later. Although a portmortem examination was

not obtained, she was thought at the time of death to have generalized sarcomatosis with cerebral involvement.

Case II. M.D.V., a white boy aged five years and ten months, first began to complain of pain in his upper left thigh about twelve weeks before referral to M. D. Anderson Hospital and Tumor Institute. This was accompanied by a limp. After two weeks the family physician was consulted, but, because the examination revealed no abnormality, the pain was attributed to mild trauma and no treatment was offered. No roentgenograms were obtained. Though intermittent limping persisted, the pain subsequently subsided only to recur six weeks prior to admission. At this time the pain was more severe, particularly early in the morning. A hard $5 \times 2\frac{1}{2}$ inch swelling without tenderness or heat had appeared in the upper lateral aspect of the left thigh. Roentgenography revealed a sclerosing lesion in the other femur, the humeri, and the sacrum. He was admitted to another hospital, where biopsy of the left femoral lesion revealed osteogenic sarcoma. At the time of the biopsy, the bony tumor at the site or excision hemorrhaged profusely, requiring emergency transfusion. He was then referred to M. D. Anderson Hospital.

Physical Examination. On admission, the child was complaining of pain in the right shoulder and left groin. The left lower limb was encased in a hip spica. The child appeared somewhat thin and undernourished, and had some small palpable cervical and inguinal lymph nodes of questionable significance.

There was no history of trauma, unusual medication or exposure to heavy metals or radioactive materials. The family and past history were not significant. The boy had been born two months prematurely, with a birth weight of 3 pounds 2 ounces, but subsequently he had developed normally.

Laboratory Findings. Prior to admission, laboratory investigation revealed a serum calcium of 10.8 mg. per cent, phosphorus 4.5 mg. per cent and alkaline phosphatase 64.6 units (Bodansky). There were no further blood chemistry studies performed. Hemoglobin on admission was 10.6 gm. per cent. The remaining blood studies and urinalysis were not abnormal.

Roentgen Findings. The initial roentgenographic study had included only the humeri, femora and pelvis, all of which were involved by sclerotic lesions (Fig. 4*A*). A complete skeletal survey at the time of admission showed predominantly osteoblastic deposits in the proximal ends of both femora, including the epiphysis on the left, both acetabula and the crest of the left ilium. The adjacent ends of both femora and tibiae were involved (Fig. 4*B*) as was the proximal end of the left fibula. Similar deposits were present in the vertebral end of the left fourth rib, the left pedicle of T-1, the body of T-6, the body of T-10, the medial ends of both clavicles and the glenoid area of the right scapula. The right wing of the sacrum was also involved. The lesions were metaphyseal in location, with the epiphyses largely, though not completely spared. The largest lesion was in the proximal portion of the left femoral shaft, the area to which the initial complaints were directed. The osteoblastic element predominated, with the lytic component relatively minor. The distribution of lesions is shown in Figure 1*B*.

Roentgenograms of the chest showed multiple densities in both lung fields, many of which appeared to contain small areas of ossification. Review of the previous chest roentgenograms showed that these had largely developed since the initial study of April 24, 1957, but tiny areas of calcification which were apparent on the earlier study may have been related to metastatic disease.

Histopathology. The original biopsy of the left femur, performed on May 2, 1957, was reviewed and disclosed neoplastic cells varying in size, shape and depth of stain, and showing great variation with respect to polarity. A few areas which appeared highly cellular were present, but, for the most part, considerable eosinophilic matrix was noted between clusters of tumor cells. Focally this osteoid matrix contained material that was interpreted to be hyaline cartilage and accompanying chondrocytes. There was also histologic evidence of extensive sclerosis.

The main problem that arose in interpretation of the lesion was whether a primary tumor had arisen in the left femur and rapidly metastasized, or whether this represented multicentric growth. Bizarre forms of hyperostosis from other causes appeared to be conclusively excluded by the histologic studies (subsequently confirmed by the clinical course and at autopsy). Consultation with the Armed Forces Institute of Pathology yielded a similar opinion. The greater involvement of the left femur might indicate origin at this site. In general, however, sclerotic

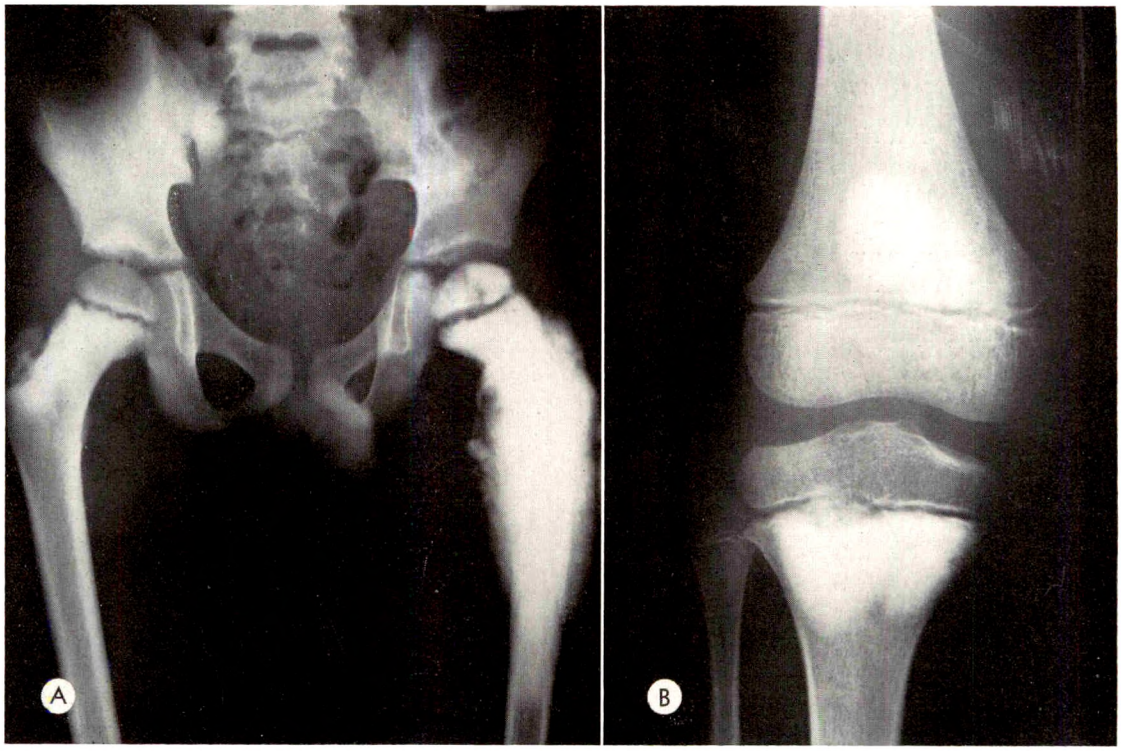


FIG. 4. Case II. (*A*) Roentgenogram shows several areas of osteosclerosis scattered throughout the pelvis, as well as extensive involvement of the proximal portion of the left femur and similar but less extensive involvement of the proximal portion of the right femur. (*B*) Roentgenogram of the right knee shows metaphyseal osteosclerosis.

osteogenic sarcomata do not rapidly metastasize and the presence of multiple bony lesions at the initial study indicates multicentric origin as a stronger possibility, though the matter is not susceptible of definite proof.

Clinical Course. The consensus was that little could be offered other than pain-relieving measures. During hospitalization the child required codeine and phenaphen 1 to 4 times daily. The cast was bivalved and the patient was discharged on analgesics. In the next four months, pain increased in severity, requiring demerol, then morphine, and, in September, 1957, the child was readmitted for alcohol block. Pain relief following the block lasted for three days.

Roentgenograms taken on the second admission (October 1, 1957) revealed new osteoblastic disease in the calvarium, base of the skull and first cervical vertebra. Most of the upper and lower dorsal vertebral bodies as well as all of the lumbar vertebrae showed similar osteoblastic disease (Fig. 5*A*). Bilateral ossifying pulmonary metastases were increasing in number (Fig. 5*B*). Hemoglobin levels had to be maintained by repeated transfusions, but the hemogram was not otherwise remarkable. Urinalysis showed a specific gravity of 1.025 with a pH of 6, but was otherwise negative.

The patient was discharged following the alcohol block but was readmitted on the fourth day because of pain. The second block was successful in relieving pain for two days and preparations were made for prefrontal lobotomy. However, prior to operation, he developed a fever of 104 degrees and respiratory distress. A chest roentgenogram at this time disclosed progressive metastatic disease with an increase in heart size and calcification of the pericardium. The diffuse bullous appearance of the heart suggested pericardial metastases with effusion.

Autopsy confirmed the presence of the multiple osteogenic sarcomata. Microscopically, the lesions were highly cellular with little intercellular osteoid matrix (Fig. 6). Tumor giant cells and bizarre mitotic figures were prominent.

COMMENT

The roentgenographic features of this

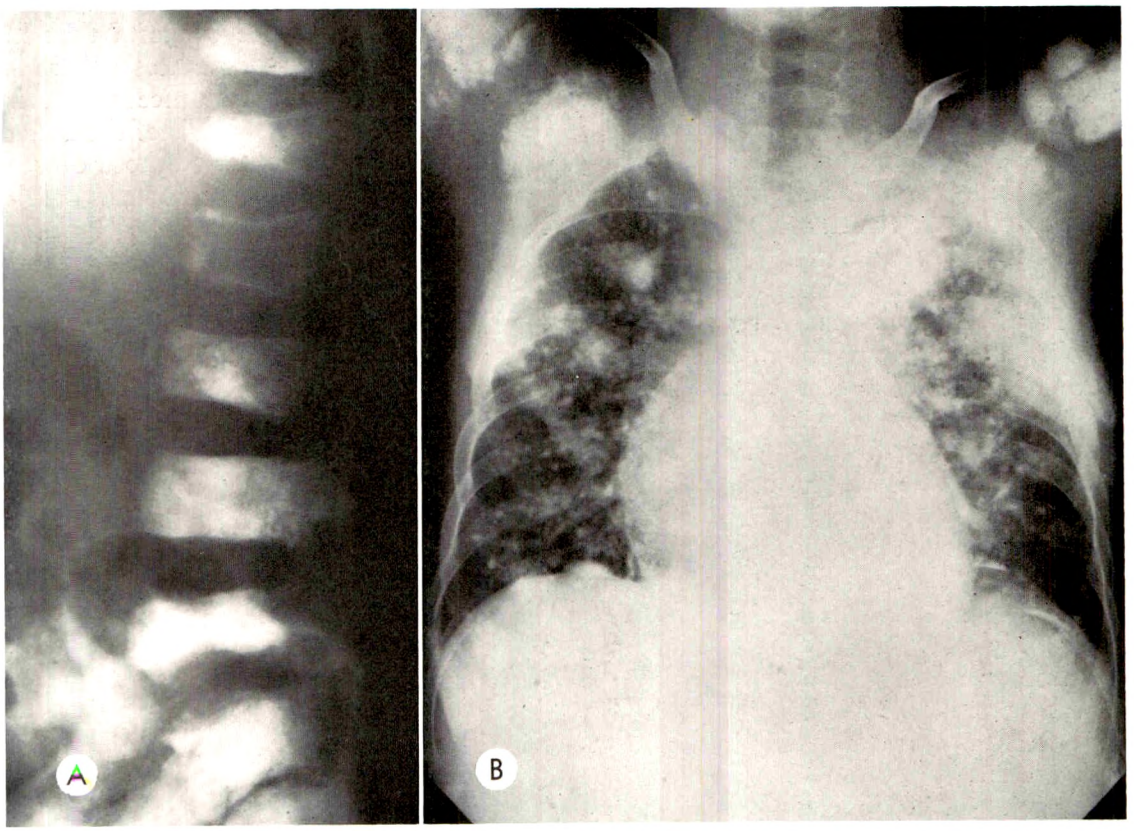


Fig. 5. Case II. (*A*) Lateral roentgenogram of the spine shows several compression fractures, as well as multiple areas of osteosclerosis. (*B*) Chest roentgenogram shows extensive osseous involvement, as well as bone-forming pulmonary metastases.

form of bone neoplasm are striking and, we believe, diagnostic.

In each of the cases previously reported, the malignancy progressed rapidly and terminated fatally. Treatment with cytoxan in Case I offered only temporary symptomatic relief with some apparent impediment in the growth of the lesions. One interesting feature observed in Case I was the presence of neoplastic tissue in the urine which was originally thought to represent metastases to the urinary tract but actually represented a cytoxan effect.

Because in Case I most of the lesions were initially of the same size and arose in the metaphyses adjacent to the epiphyseal line, they are believed to have arisen from multicentric foci rather than to represent osseous metastases with or without transpulmonary spread. As far as the second case is concerned, the disparity in size between the

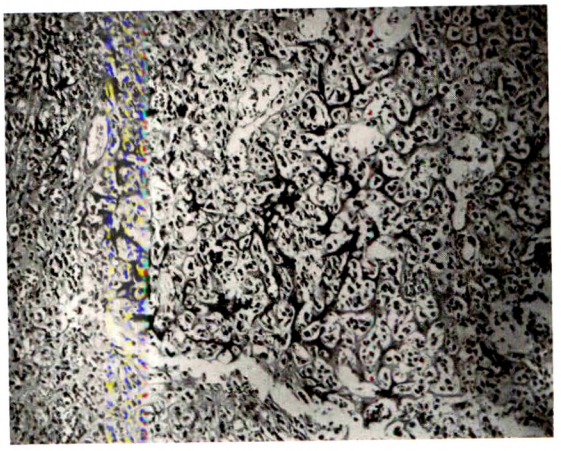


Fig. 6. Case II. Photomicrograph shows nests of dense cellularity which are separated by thin strands of eosinophilic osteoid matrix. There is considerable variation in size and shape of the individual cells.

lesion in the femur and those seen in the other bones raises a question as to the category in which it belongs. Despite this, there is a fairly strong presumption of coincident origin in several bones, and, in any case, its behavior and appearance are similar to that described in sclerosing osteogenic sarcomatosis.

The over-all appearance is strikingly similar to multiple bone sarcomas which arise from the osseous deposition of certain radioactive substances such as radium, strontium and beryllium. No evidence for such an etiology has been presented in any of the cases. The possibility of a toxic factor being responsible for the generalized conversion of normal endochondral bone formation to a malignant process must be considered.

The high serum alkaline phosphatase values in these cases are typical for bone lesions with marked osteoblastic activity. As noted histologically, new bone formation was irregular. Bone itself is probably the result of the local increase in enzyme rather than a cause. The persistent normal values for serum calcium and phosphorus suggest that the degree of new bone formation was insufficient to be reflected in this area. At one time in Case 1, after institution of the chemotherapy, the serum alkaline phosphatase dropped, although it was still abnormally elevated. This temporary reduction in enzyme concentration has also been described after surgical removal or therapeutic irradiation of a single tumor.[2]

In the differential diagnosis one should consider those conditions which produce localized areas of osteosclerosis. The condition which may appear roentgenographically identical to sclerosing osteogenic sarcomatosis is osteogenic sarcoma secondary to the ingestion of radioactive heavy metals, notably radium, phosphorus or strontium. In these cases there are dense areas of sclerosis localized to the metaphysis adjacent to the epiphyseal lines, as well as evidence of periosteal elevation and extension of the malignant process into the surrounding soft tissues. The similar roentgenographic appearance suggests that in sclerosing osteogenic sarcomatosis there is an underlying toxic substance which is deposited in the growing ends of the bones producing changes similar to those caused by the heavy metals. Differentiation is made by failing to find evidence of heavy metal poisoning by laboratory methods and by history as well as by failure to detect evidence of radioactivity at the sites of involvement. Other conditions of localized osteosclerosis such as sclerosing osteitis, progressive diaphyseal dysplasia (Engelmann's syndrome), hereditary diaphyseal sclerosis (Ribbing's syndrome), melorheostosis, osteopoikilosis, bone infarction and some forms of lymphoma are characteristic in their roentgenographic appearance and do not show the combined features of sclerosis and malignancy as demonstrated in sclerosing osteogenic sarcomatosis. Osteopetrosis produces a more generalized form of osteosclerosis and should not be confused with this condition.

SUMMARY

Two cases of multiple osteogenic sarcomata in children are presented. The roentgenographic findings are diagnostic and the distribution and size of the sclerotic lesions strongly suggest a multicentric origin.

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SCLEROSING OSTEOGENIC SARCOMA*

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OSTEOBLASTIC or sclerosing osteogenic sarcoma with involvement of multiple skeletal sites is an infrequently encountered entity. Sporadic case reports have appeared in the English literature. Some difference of opinion exists regarding the pathogenesis of the disseminated bony lesions in a sclerosing type of osteogenic sarcoma. Whether these foci represent metastatic deposits or multiple primary lesions is an unsettled question. The following case is presented because it typifies the characteristic roentgen appearance of this neoplasm and has features which may help clarify the question of etiology of the disseminated bony lesions.

REPORT OF A CASE

The patient was a 6 year old white female who had been well until November 4, 1960, when her mother noted a swelling of the left upper arm. On November 9, roentgen examination of the left humerus revealed a 7×3 cm., well circumscribed mass of new bone formation in a juxtacortical location on the posterior surface of the proximal metaphysis (Fig. 1). This had the appearance of an extrusion of bone through a break in the cortical surface. In limited areas there was perpendicular radiation of bone spicules. Periosteal elevation with subperiosteal new bone formation parallel to the shaft was also apparent. Amorphous deposits of bone were noted extending the length of the humeral shaft and the epiphysis contained dense bone deposits. A roentgenogram of the chest showed small, partially calcified foci disseminated throughout the lung fields and involving the pleura (Fig. 2). The initial impression was that this represented either a sclerosing type of osteogenic sarcoma with pulmonary metastases, or a very low grade osteomyelitis with calcified parenchymal granulomata. Fungus infection was also considered to be a possible etiology, particularly in view the clinically healthy appearance of the patient.

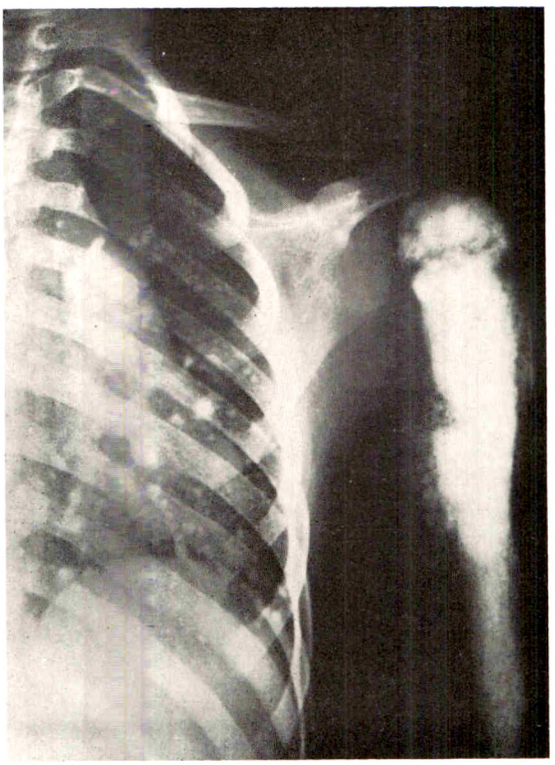


FIG. 1. Sclerosing type of osteogenic sarcoma, manifested by a juxtacortical mass of new bone formation, periosteal elevation and central medullary deposits.

The child was admitted to Children's Hospital of Michigan on November 9, 1960. The initial physical examination revealed an apparently well child except for the presence of a hard, slightly tender mass in the posterior medial aspect of the upper one-third of the humerus. The chest was clear to auscultation and percussion.

Laboratory examinations revealed: hemoglobin 12.5 gm. per 100 ml.; white blood cell count 3,000 per cubic millimeter; differential count—polymorphonuclears 40 per cent; lymphocytes 6[illegible] per cent; mononuclear lymphocytes 4 per cent; and eosinophils 4 per cent. The platelets were adequate on the peripheral

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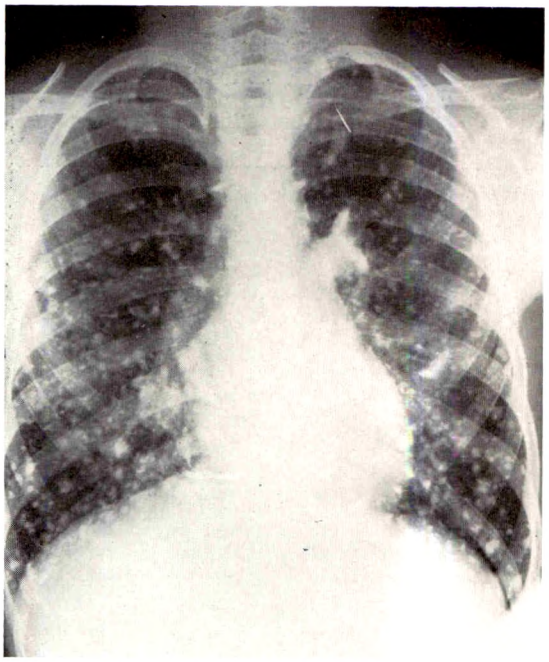


FIG. 2. Chest roentgenogram shows deposits of calcific density disseminated throughout the lung fields and pleura.

smear. Serum calcium was 12.6 mg. per 100 ml., phosphorus was 3.6 mg. per 100 ml., the alkaline phosphatase was 33.4 Bodansky units and the urinalysis was negative. A Sulkowitch test was not done.

Skin tests for histoplasmosis, tuberculosis (PPD's in dilutions of 1:1,000 and 1:500), coccidioidomycosis and blastomycosis were reported as negative.

On November 12, 1960, a bone survey revealed limited zones of sclerotic density involving the right iliac crest, left sacrum and proximal metaphysis of the left fibula. The skull and other long bones were negative.

On November 15, 1960, an open biopsy of the left humerus was performed. The periosteum was stripped easily from the bulky tumor mass which was marble hard and solid. Specimens were taken from the humeral shaft.

Histologic examination of the biopsy specimen showed irregular cellular tissue in which the cellular elements had an irregular, sometimes bizarre and frankly neoplastic appearance (Fig. 3). Only a few areas of the tumor were fibrous and the bulk of it consisted of large masses and deposits of osteoid which appeared to have been formed by the tumor cells. In many areas the osteoid had undergone ischemic necrosis and was hypocellular.

The postoperative course was uneventful and the patient was discharged on November 20, 1960.

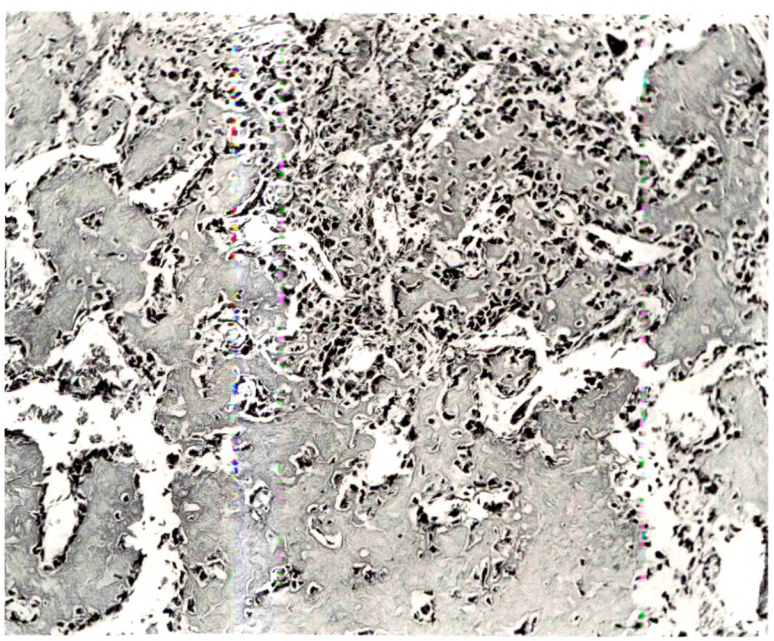


FIG. 3. Histologic section of the primary tumor shows the production of large amounts of osteoid. The cellular elements are pleomorphic and irregularly distributed in the tumor. (Hematoxylin and eosin stain, × 120.)

Repeat roentgen examinations performed on December 21, 1960, and February 8, 1961, revealed progression of the previously described areas of involvement with confluence of the pulmonary lesions. New foci of bone formation were noted in the left lateral femoral condyle (Fig. 4), left and right ilia (Fig. 5), right femoral neck and right capital femoral epiphysis, as well as in the left scapula.

The patient became increasingly dypsneic and expired on May 13, 1961. Postmortem examination showed tumor tissue completely filling the ossification center of the epiphysis, as well as the shaft of the left humerus. The periosteum was also extensively infiltrated and a large external tumor was present on the medial side of the bone. The presence of tumor tissue was confirmed in the pelvic bones, dorsal spine, left scapula, femora and ribs. Metastatic tumor was present in the lungs and pericardium, and tumor tissue also infiltrated the diaphragm and the left lateral chest wall.

DISCUSSION

Lindbom *et al.*,[6] in a review of 92 cases of osteogenic sarcoma, state that a diagnosis could be made roentgenographically in 64 per cent of the cases. Once familiar with the distinctive characteristics of the sclerosing type of osteogenic sarcoma, as described in this report, the radiologist should be able to render a correct diagnosis in a high percentage of cases. Biopsy is necessary for confirmation, however.

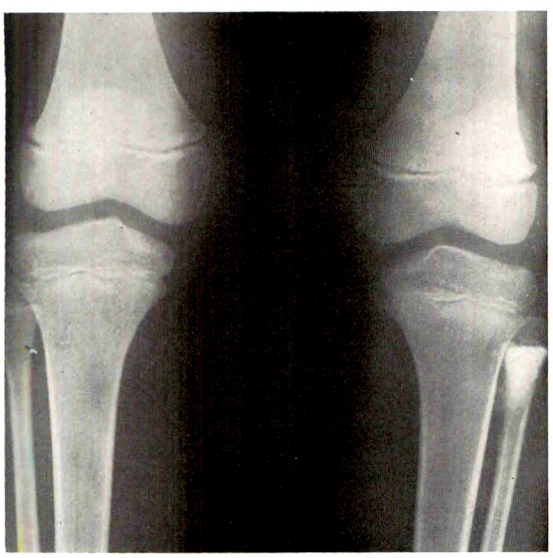


Fig. 4. Sclerotic lesions are present in the metaphyses of the left distal femur and proximal fibula. Periosteal reaction associated with the fibular tumor is evident.



Fig. 5. Roentgenogram shows the appearance of secondary sites in the pelvis and femora.

The roentgen features of this case are typical of those described by other authors.[4,8] The primary site of involvement is usually the metaphysis of the long bones, with marked bone production and minimal bone destruction. The tumor may be confined by the cortex but usually breaks through the cortex and even the periosteum to form a bony mass in the juxtacortical region. It extends through the medullary cavity and is usually confined in this direction by the articular cartilage in the adult and the epiphysial plate in the juvenile. The scattered areas of bone involvement may exhibit a similar appearance but are frequently central medullary deposits of sclerotic bone without overlying cortical disturbance or periosteal reaction. Initially only an ill defined sclerotic density may be manifest.

Geschickter and Copeland[4] describe the epiphysial plate as being a most effective barrier to the advance of sarcoma. Jaffe[5] states, however, that tumor cells can extend across the epiphysial plate.

Roentgen examination has revealed epiphysial involvement in some cases,[3,8] but no pathologic studies are cited. Roentgenographically, the ossification center of the

epiphysis at the head of the humerus in this case appeared to be densely replaced by tumor. Pathologic study of the epiphysial plate was performed to determine the integrity of this structure. Random sections through the cartilaginous epiphysial plate showed that it was perforated in several places by tumor tissue extending through these areas (Fig. 6). The presence of pulmonary metastases is a variable finding. In Moseley and Bass's[8] report, no pulmonary lesions were described. Badgley and Batts,[1] in a review of 80 cases of osteogenic sarcoma, refer to the pulmonary metastases but make no mention of multiple bony sites in the sclerotic type. The pulmonary lesions associated with the sclerotic type of osteogenic sarcoma are frequently densely calcific and have been shown to represent heterotopic bone formation by metastatic osteoid tissue, rather than calcium deposition in necrotic tumor tissue.[12]

In 1936, Silverman[11] reviewed the literature and concluded that the multiple bony sites in this type of sarcoma arise in the periosteum. It was thought that they represented multiple primary tumors, rather than metastatic foci, although this problem is still unresolved. The bone lesions in multiple myeloma are generally believed to represent multifocal sites of origin. The presence of disseminated osseous sites in Ewing's sarcoma is attributed to metastatic spread. The work of Batson,[2] in demonstrating the anatomic communications and physiologic function of the vertebral system of veins, provides a most logical reason for also considering these scattered osseous lesions of osteogenic sarcomata as metastases. That communication of venous vessels from the bones of

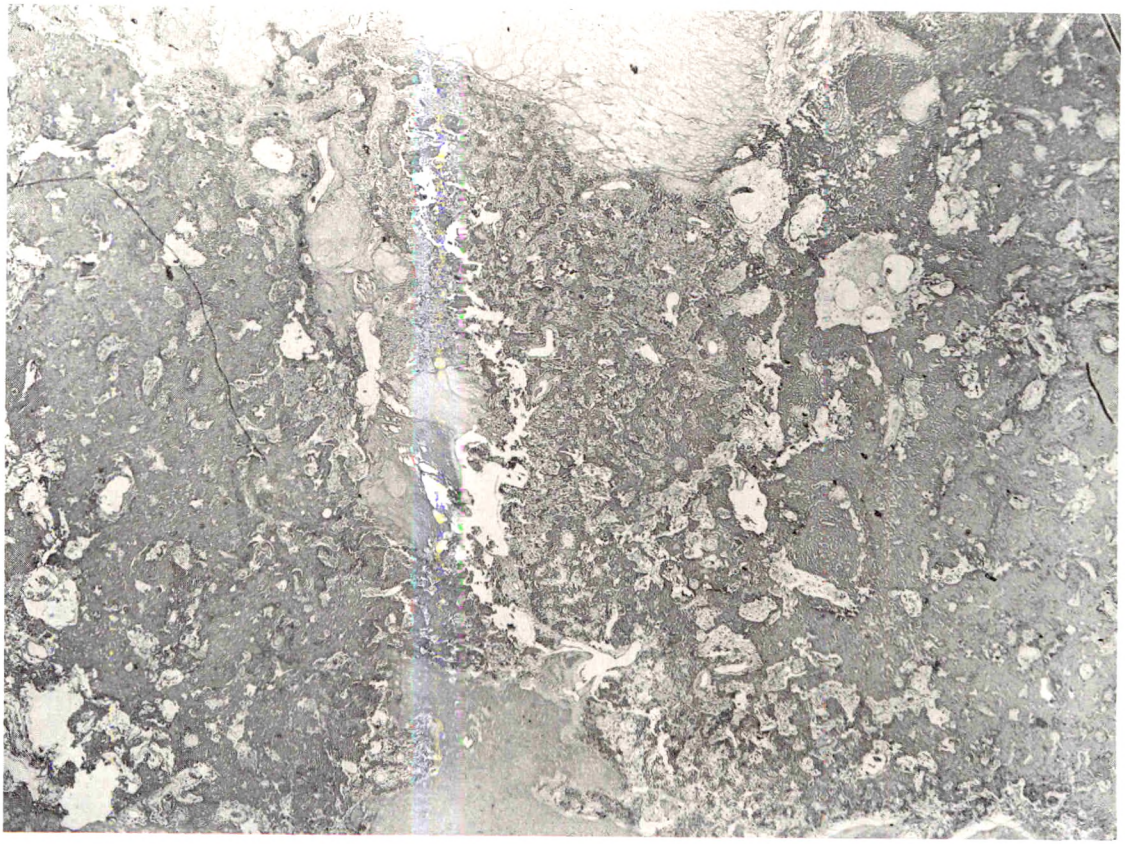


FIG. 6. Tumor extends from the metaphysis into the proximal epiphysial ossification center of the humerus through a gap in the cartilaginous plate. (Hematoxylin and eosin stain, × 10).

the extremities with this vertebral system occurs is amply documented by Perey *et al.*,[9] and Batson.[2] The ebb and flow of this blood within the vertebral system carries blood in ascending and descending fashion along the paravertebral plexus, and proximally and distally in the smaller valveless veins of the extremities without passing through the heart or lungs except by communication with the inferior vena caval or superior vena caval system. Other neoplasms have been shown to metastasize by this route.[7] The frequency of neoplastic deposits in the spine and pelvis in cases similar to ours suggests dissemination by the vertebral system. The lack of pulmonary metastases has been pointed out as evidence in favor of disseminated primary foci.[10] The presence or absence of pulmonary lesions is explained by the venous route taken by the cells. Batson states, "Of course, the ultimate direction of flow is toward the heart, and the secondary lesions could be in the lungs. The point is they need not be in the lungs since the vertebral system bypasses the portal, the caval and pulmonary systems."[2]

The differential diagnosis of these lesions includes parosteal sarcoma. This usually occurs beyond the age of 30 years, is juxtacortical, without medullary involvement, and usually solitary but may metastasize to the lungs.

Ewing's sarcoma may rarely occur in an exclusively sclerotic form and can metastasize to lung and bone. It frequently has a laminated appearance but biopsy may be the final determinant.

Although osteomyelitis was initially considered in this case, it would be most unusual to have an entirely osteoblastic type of reaction to infection, mycotic or bacterial, especially in its acute phase. The character of the disseminated lesions also militates against this diagnosis.

SUMMARY

Sclerosing osteogenic sarcoma can occur in multiple bony sites and has a characteristic roentgen appearance. That invasion of the epiphysis by migration of malignant cells across the epiphysial plate can be found is demonstrated pathologically. The hematogenous dissemination of neoplastic cells by the vertebral plexus of veins, in patients who do not have pulmonary metastases, as a possible explanation for the disseminated bony lesions is discussed. Although final proof is lacking, it is our belief that these bony sites represent metastatic deposits rather than multifocal primary tumors.

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We are indebted to Robert H. Burge, M.D., for permission to report this case.

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SCAPULAR LESIONS IN CHILDHOOD*

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PHILADELPHIA, PENNSYLVANIA

LESIONS of the scapula in childhood are rare and the resulting disturbance in the bone architecture is often not diagnostic of the underlying condition. Different disease entities such as inflammation, reactive bone processes (*i.e.*, infantile cortical hyperostosis) and benign and malignant tumors may present a similar roentgen appearance. At times the correct diagnosis is achieved with the help of clinical and laboratory findings together with roentgen changes in other parts of the body.

In recent years we have seen 10 cases of primary lesions of the scapula (Table 1). These 10 cases represent 6 different disease processes and an example of each is given. Some of these lesions were correctly diagnosed by clinical and roentgen findings, while others required biopsy and pathologic examination.

REPORT OF CASES

CASE 1 (Fig. 1, *A* and *B*). G.J., a 7 week old female, was admitted to the hospital because of inability of 1 week's duration to move the right arm. Some tenderness was elicited over

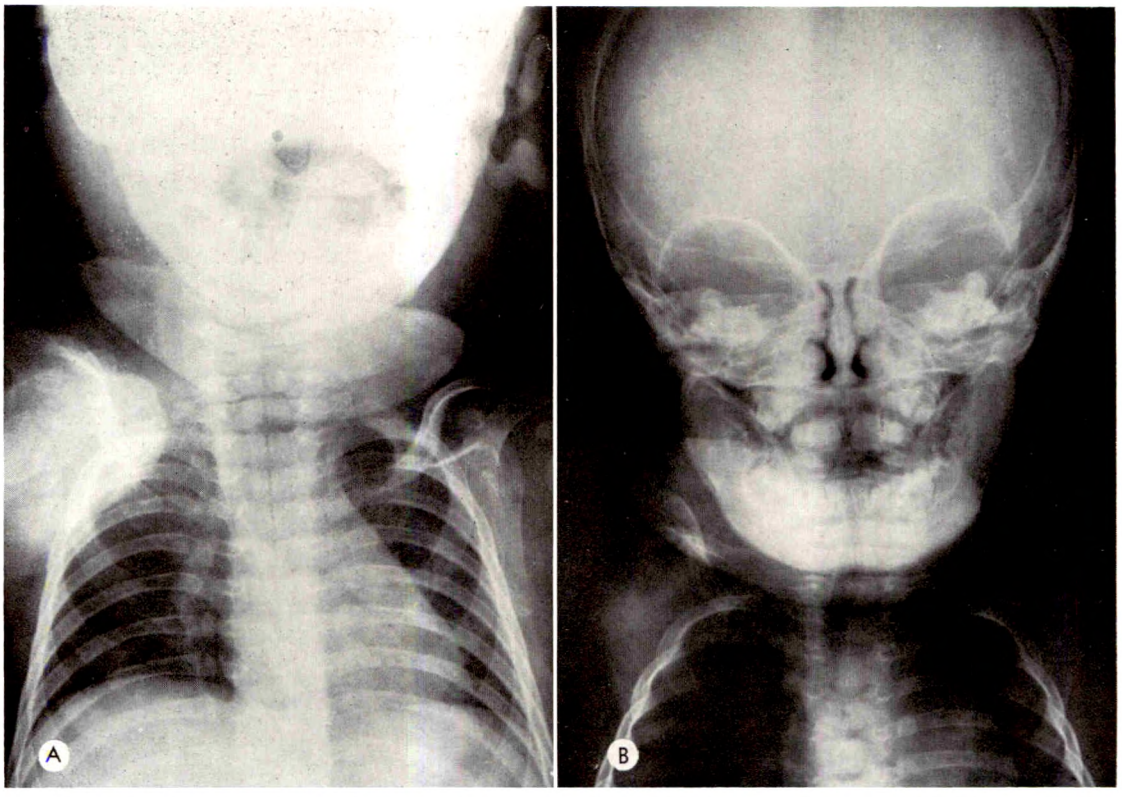


FIG. 1. Case 1. (*A*) Chest roentgenogram showing reactive changes in the right scapula and mandibular cortical thickening. (*B*) Anteroposterior view of the mandible 3 months later showing increased mandibular cortical thickening. The scapula shows less reactive change.

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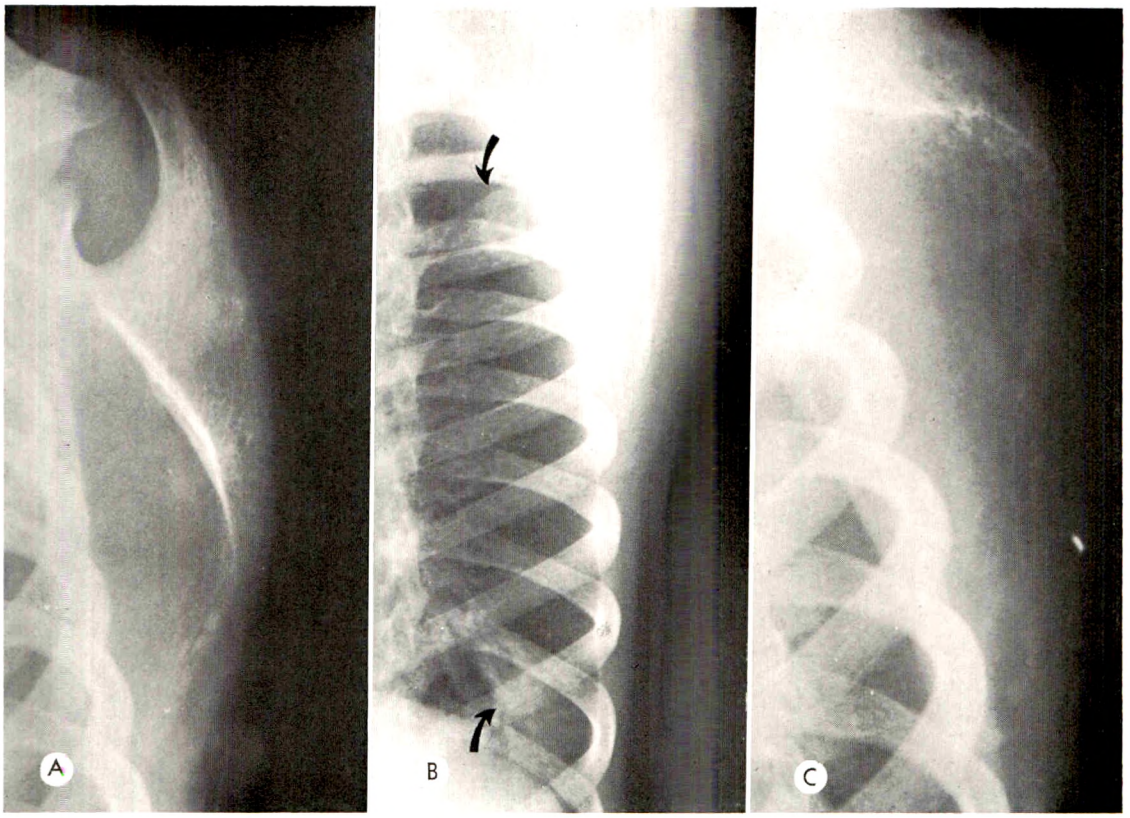


Fig. 2. Case II. (*A*) Oblique view of the left scapula showing soft tissue edema and minimal demineralization. (*B*) Left anterior oblique view of the left chest showing infiltration in the upper and lower lung field (arrows). (*C*) Oblique view of the left scapula 10 days after biopsy showing bone destruction. A silver clip marks the biopsy site.

the posterior aspect of the right shoulder. The baby was irritable, and had a temperature of 99°F. On close inspection some edema of the face was noted. Roentgen examination of the right shoulder (Fig. 1*A*) revealed extensive osteoblastic changes of the right scapula. Cortical thickening of the mandible was also noted. The diagnosis of Caffey's disease was therefore established. Three months later the mandibular cortical hyperostosis was more extensive (Fig. 1*B*), but the scapular lesion had regressed.

Case II (Fig. 2, *A*, *B* and *C*). N.G., a 10 year old female, was admitted because of pain of 2 weeks' duration in the left shoulder. Four weeks prior to admission she fell and struck her left shoulder, but it was not until 2 weeks later that she experienced a sudden onset of pain, swelling, and stiffness of the shoulder. The temperature on admission varied from 100° to 105°F. A diffuse swelling over the left supra-scapular region was noted, together with a faint erythema of the skin. Definite limitation of motion of the left shoulder was present. The white blood cell count was 22,300 with 70 per cent polymorphonuclear leukocytes and the sedimentation rate was 30 mm./hr. (Cutler). Roentgen examination of the left shoulder showed soft tissue edema over the scapula with some demineralization (Fig. 2*A*). A chest roentgenogram revealed some infiltration of the left upper and lower lung fields (Fig. 2*B*). A

Table I

Scapular lesions occurring in childhood

Infantile cortical hyperostosis	3
Eosinophilic granuloma	2
Staphylococcal osteomyelitis	2
Aneurysmal bone cyst	1
Lymphangioma	1
Reticulum cell sarcoma	1
Total	10

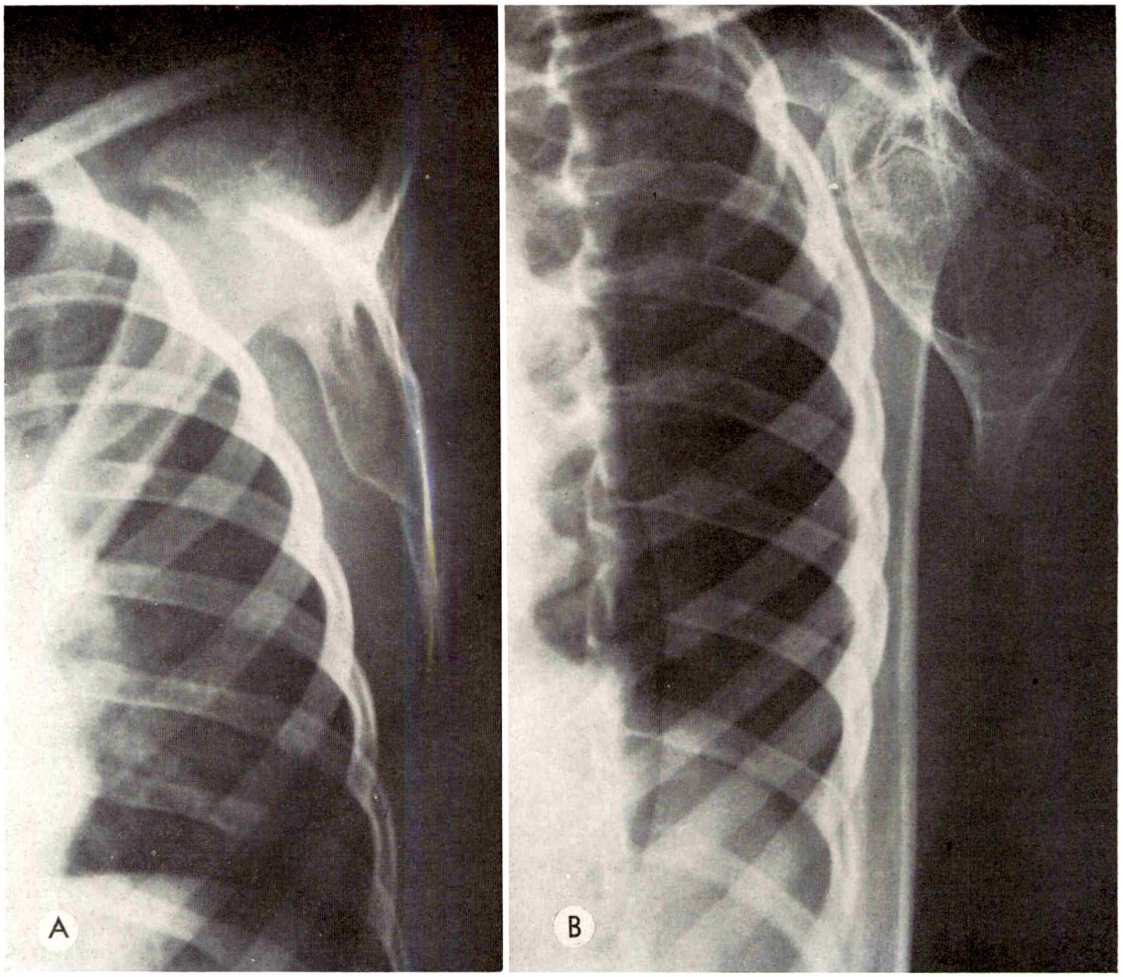


FIG. 3. Case III. (*A*) Oblique view of the left scapula showing an expansile cystic lesion with a fracture through the anterior cortex. (*B*) Oblique view 1 year later showing progression of the cystic lesion.

biopsy of the left scapula was performed 10 days after admission, and a diagnosis of staphylococcal osteomyelitis was established. A roentgen examination of the left scapula 10 days after biopsy showed extensive bone destruction (Fig. 2*C*). The patient was treated with erythromycin and made a complete recovery. Roentgenographically, the scapula presented a normal appearance.

CASE III (Fig. 3, *A* and *B*). M.T., a 7 year old female, was admitted because of a painful left shoulder of 1 week's duration following a fall. On physical examination tenderness and swelling were noted over the left scapula. Roentgen examination of the left shoulder showed an expansile cystic lesion of the left scapula with a fracture through the anterior cortex (Fig. 3*A*). Biopsy was performed and a diagnosis of an aneurysmal bone cyst was established. The lesion was thoroughly curetted. One year later there was roentgen evidence that the lesion had caused further bone expansion (Fig. 3*B*). A second more extensive curettage as well as packing with bone chips was performed. Two years later the scapula presented an almost normal architecture.

CASE IV (Fig. 4, *A* and *B*). E.M., a 4 year old male, was admitted because of a mass of 1 day's duration in the region of the right scapula. While giving the boy a bath, the mother noted a lump in the region of the right shoulder. On physical examination a nontender mass was palpable overlying the right scapula. Roentgen examination (Fig. 4, *A* and *B*) revealed an

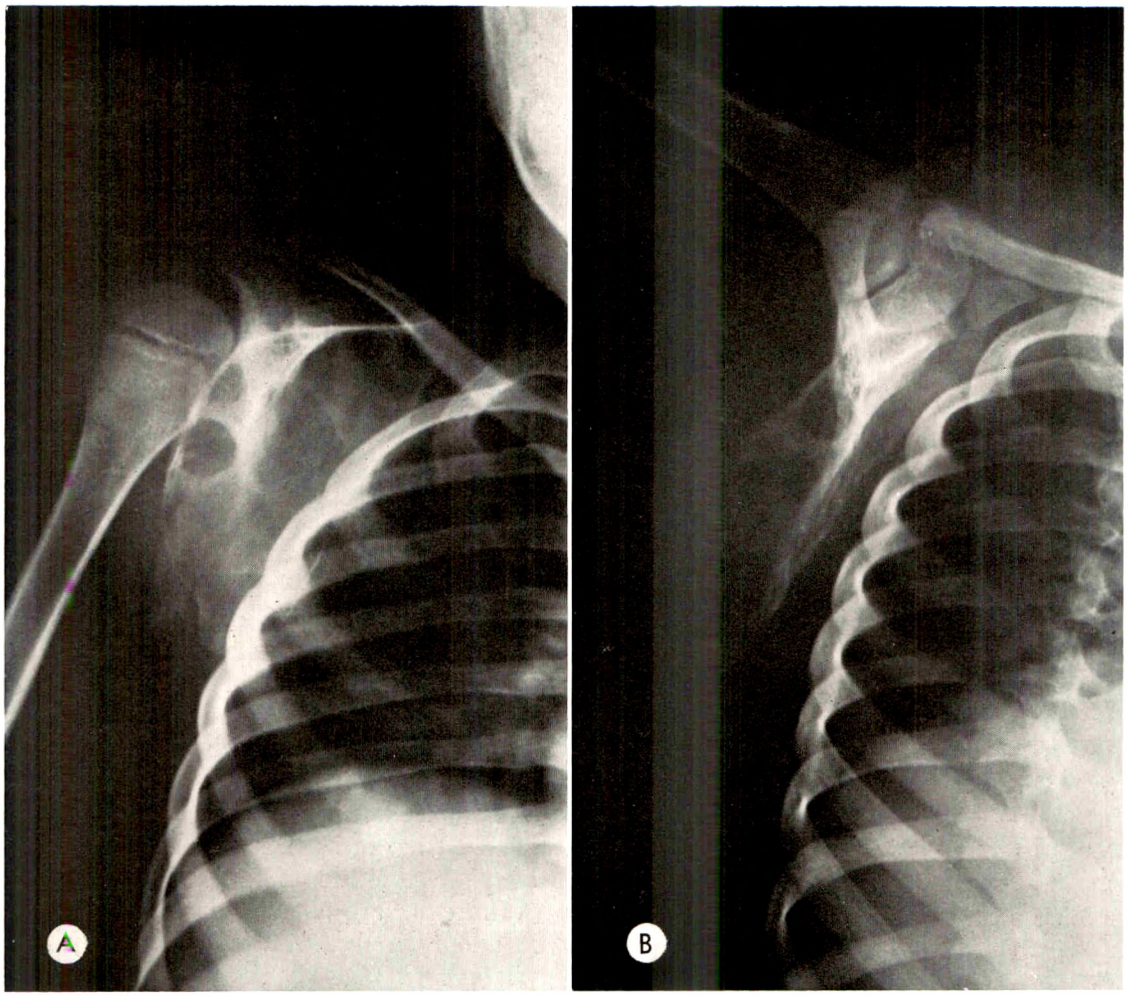


Fig. 4. Case iv. (*A*) Anteroposterior view of the right shoulder showing a multiloculated expansile cystic lesion of the right scapula. (*B*) Oblique view of the right scapula showing a fracture through the posterior cortical wall. Note also the overlying soft tissue density.

expansile multicystic lesion of the entire body of the scapula with a fracture through the posterior cortex. Considerable soft tissue swelling was present over the scapula. A biopsy was performed the following day and a diagnosis of eosinophilic granuloma was established. The patient received 600 r (air) in 6 days. The lesion of the scapula healed without further treatment.

Case v (Fig. 5). W.R., a 13 year old male, was admitted to the Hospital of the University of Pennsylvania because of a painful right shoulder of 6 months' duration following a fall. The pain had been intermittent and seemed localized over the anterior aspect of the shoulder, On physical examination tenderness was elicited over the anterior right shoulder, the acromion process, and the lateral third of the spine of the scapula. Roentgen examination of the right shoulder showed a multiloculated cystic expansile lesion of the right scapula (Fig. 5). Biopsy was performed and a diagnosis of lymphangioma was established. After initial biopsy and thorough curettage, the lesion recurred within a period of 1 year. A second operation, consisting of total local excision and bone graft, was carried out. Follow-up studies over a period of 18 months showed complete healing without evidence of recurrence.

Case vi (Fig. 6, *A*, *B*, *C* and *D*). C.C., a 4 year old female, was admitted because of pain in the right shoulder of 9 days' duration. The pain began following an injury to the right shoulder. Four days prior to admission the

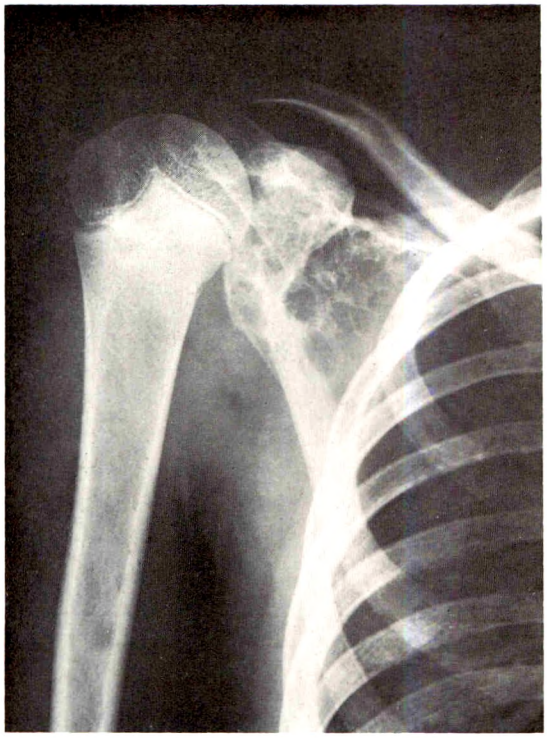


FIG. 5. Case V. Anteroposterior view of the right shoulder showing a multiloculated expansile cystic lesion of the scapula.

mother noted a lump overlying the right shoulder. On physical examination, a nontender mass was palpated over the right scapula. Roentgen examination (Fig. 6, *A* and *B*) showed considerable soft tissue swelling over the right scapula with some cortical destruction. Biopsy was performed and a diagnosis of a reticulum cell sarcoma was established. The lesion was treated by roentgen therapy with 4,000 r delivered to the tumor in 3 weeks. Re-examination in 1 month showed a definite improvement roentgenographically (Fig. 6*C*). Four months later there was evidence of extensive bone metastases (Fig. 6*D*), and the child expired 6 months after the initial admission.

DISCUSSION

In evaluating the cases presented, it can be seen that a correct diagnosis can sometimes be made from the clinical and roentgen findings alone. In the case of infantile cortical hyperostosis (Case I), the initial involvement of the mandible pointed to the etiology of the extensive reactive scapular changes. In another case of Caffey's disease in our series, the changes in the scapula preceded the mandibular involvement, and a biopsy was required to rule out a malignant lesion. In Caffey's original article on this entity,[1] 4 cases were reported, with the third and fourth cases demonstrating scapular involvement.

In the case of staphylococcal osteomyelitis (Case II), the presence of pulmonary lesions suggested an inflammatory process as the causative factor. The initial soft tissue swelling over the scapula without evidence of bone involvement, followed in 2 weeks by definite evidence of bone destruction, outlined the course that one would expect in osteomyelitis.

More often the diagnosis could not be made until the biopsy had been performed. In the case of eosinophilic granuloma (Case IV), a loculated expanded appearance of the scapula was present, but no other skeletal lesions were seen. In another case of eosinophilic granuloma of the scapula, the presence of a solitary radiolucent skull lesion helped to suggest the diagnosis. When the roentgenographic findings of eosinophilic granuloma[3] are compared with those seen in aneurysmal bone cyst (Case III),[2] lymphangioma (Case V),[4] and reticulum cell sarcoma (Case VI), it is obviously not possible to make an etiologic diagnosis from the roentgen appearance alone.

The prognosis of these 6 entities makes the correct diagnosis imperative. Patients with osteomyelitis should recover with antibiotic therapy. Our 2 cases of eosinophilic granuloma have been free of disease for 6 and 15 years respectively following roentgen therapy of the primary lesions. Our patients with Caffey's disease have all had spontaneous regression of the bone changes. The patient with reticulum cell sarcoma had temporary regression of the local scapular involvement after roentgen therapy but died within a few months from disseminated disease. The patients with aneurysmal bone cyst and lymphangioma both had local recurrence requiring further surgical intervention.

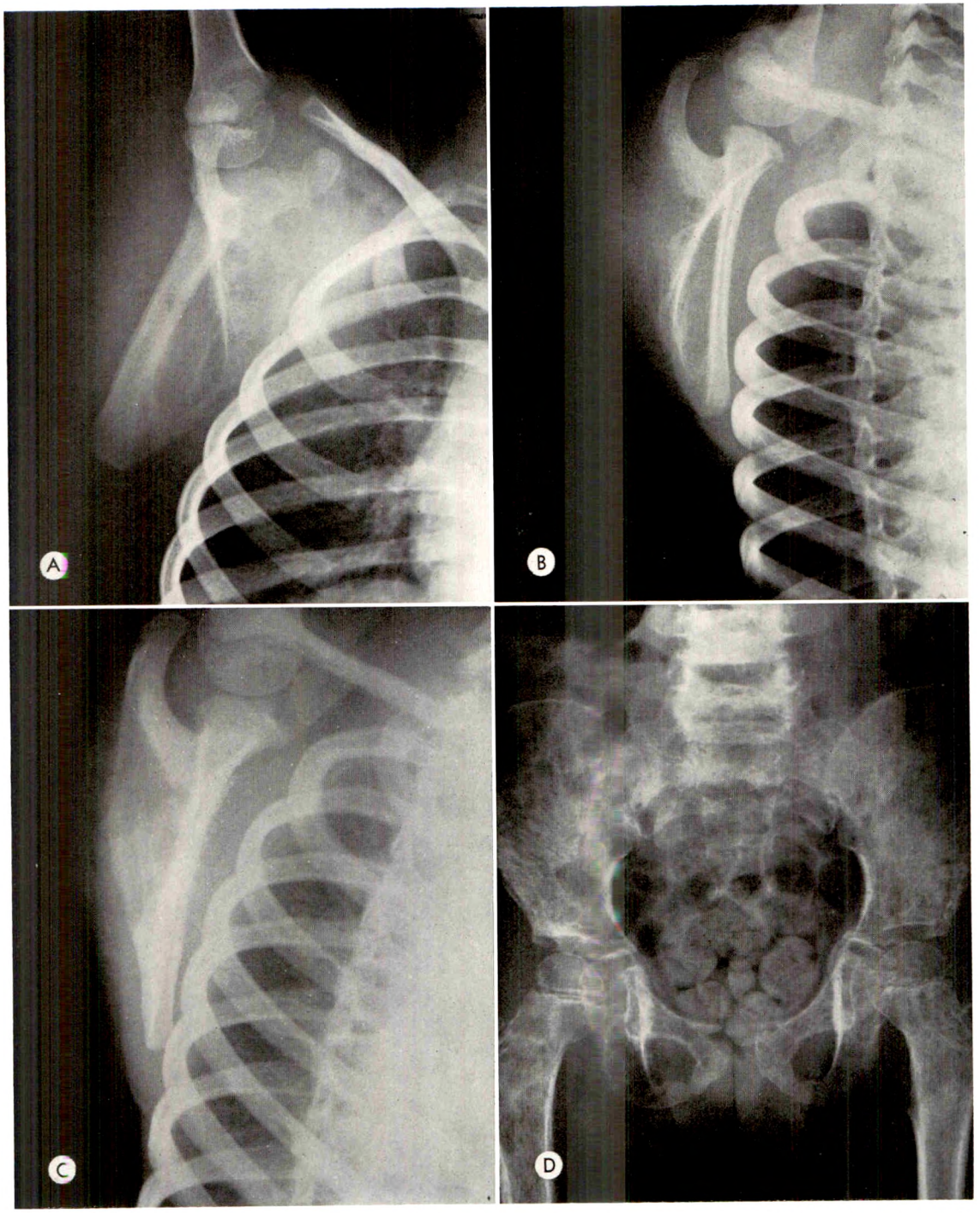


Fig. 6. Case vi. (*A*) Anteroposterior view of the right shoulder showing osteolytic lesions throughout the body of the right scapula. (*B*) Oblique view of the scapula showing considerable overlying soft tissue swelling with extensive cortical destruction. (*C*) Oblique view of the scapula 1 month after roentgen therapy showing less soft tissue swelling and apparent healing of the scapular lesion. (*D*) Anteroposterior view of the pelvis 4 months later showing widespread osteolytic metastases.

SUMMARY

Lesions of the scapula in childhood are rare. Different disease processes may cause the scapula to react in such a way that there is a similar roentgenographic appearance. The correct diagnosis can sometimes be made on the basis of clinical, laboratory and roentgen findings, but more often biopsy and pathologic examination are needed. It is important to be aware of the different disease entities that may cause roentgenographic changes in the scapula, as the management and prognosis of the individual case hinges on a correct diagnosis.

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We would like to thank Dr. E. R. Schmidt of the Department of Orthopedic Surgery, Hospital of the University of Pennsylvania, for permission to include Case v.

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HEMOGLOBIN S-C DISEASE*

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THE concept of the chronic hemolytic anemias associated with sickling erythrocytes has been altered in the past ten years. The development of hemoglobin electrophoresis has resulted in the discovery of a family of disease entities that previously had been included under the general classification of sickle cell anemia. The statistically most important of the variants of sickle cell anemia is hemoglobin S-C disease. This "new" disease explains the clinical picture of the nonanemic and elderly "sickle cell anemia" patients and aids in the identification of other manifestations that were not thought to be associated with sickling erythrocytes.

Sickle cell anemia was first described by Herrick[9] in 1910, and since that time the manifestations of the disease, both clinical and roentgenographic, have been reported often in the literature. In 1949, Pauling and his associates[18] made a major contribution to the understanding of sickling erythrocytes when they developed hemoglobin electrophoresis and made the hemoglobinopathies a molecular disease. It was now possible to identify a specific substance that was associated with the sickling process—hemoglobin S. Shortly afterwards, Itano[12] described a new hemoglobin, hemoglobin C, that, in combination with hemoglobin S, produced a similar disease complex—hemoglobin S-C disease. Many other hemoglobin types have been identified since then. The differentiation of the pertinent hemoglobin types is quite specific, as shown by the electrophoretic pattern in Figure 1. To date, the only electrophoretically identifiable hemoglobin type of major importance that, in combination with hemoglobin S, produces a "sickle cell anemia" complex is hemoglobin C. Other variants of sickle cell anemia have been noted, but are rare in occurrence. Hemoglobin S also combines with the thalassemia gene (electrophoretically hemoglobin A) to produce a severe disease complex: hemoglobin S-thalassemia.[3]

GENETICS

The hemoglobinopathies follow a mendelian dominant inheritance pattern.[3] Various genetic combinations that can produce a hemoglobin S-C pattern in a patient are shown in Figure 2. From this illustration it is seen that each parent contributes one-half of his genotype to the offspring. In all cases of hemoglobin S-C disease where family pedigrees were outlined, it was found that one parent contributed hemoglobin S and the other parent hemoglobin C to the patient. This genetic crossing produces a genotype of hemoglobin S-C, but the patient does not necessarily present with clinical symptoms. These patients are found only on electrophoretic surveys of the population.

Hemoglobin C in combination with itself (hemoglobin C-C) or in combination with hemoglobin A (hemoglobin A-C) presents essentially an asymptomatic picture.[3] Sickle cell trait (hemoglobin A-S) is well known for its benign course. Sickle cell anemia is homozygous for the hemoglobin S gene and genetically is hemoglobin S-S.

INCIDENCE

The incidence of hemoglobin S in the Negro population is about 9 to 10 per cent and the occurrence of sickle cell anemia is about 1 to 2 per cent. The incidence of hemoglobin C is 2 per cent and it is estimated, at present, that the ratio of the occurrence of hemoglobin S-C disease to sickle cell anemia is 1–3; *i.e.*, in about 0.3 to 0.7 per cent of the Negro popula-

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Presented at the Sixty-second Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 26–29, 1961.

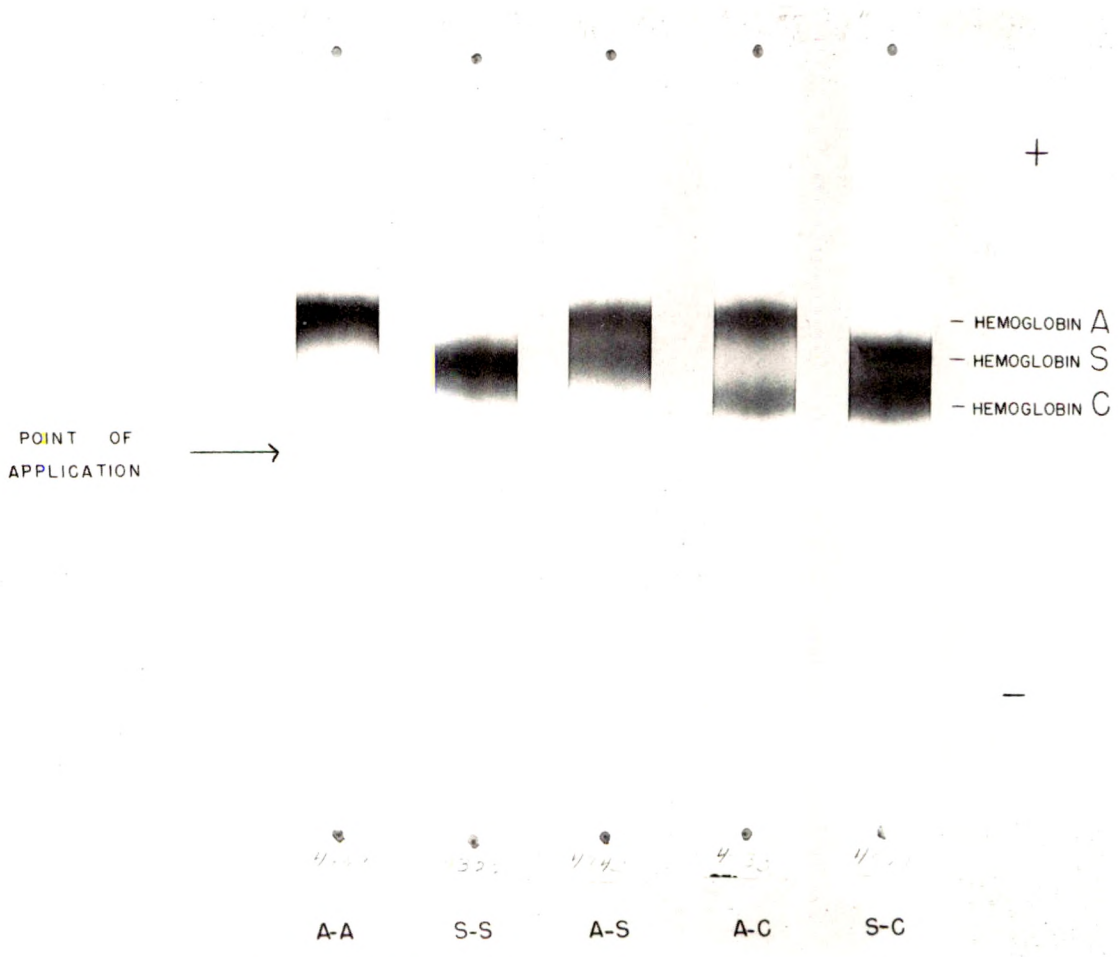


FIG. 1. The separation of the hemoglobin types is done by paper electrophoresis. The electrical charge of the globin fraction of the hemoglobin determines the migratory rate of the various hemoglobin types. This method cannot separate all the hemoglobin types (two hemoglobins may have the same migratory rate) but hemoglobin A, C and S can easily be differentiated.

tion.[3] Although this is a relatively small percentage, it represents a large number of persons and is quite significant.

PATHOLOGY

When erythrocytes containing hemoglobin S are subjected to a low partial oxygen pressure or a high partial carbon dioxide pressure, the globin portion of the hemoglobin undergoes a complex physiochemical reaction and produces a sickle erythrocyte. The degree to which this process develops is proportional to the amount of hemoglobin S present within the hemoglobin molecule.[4,22]

The sickle cell is more labile to mechanical trauma than a normal erythrocyte and is easily ruptured. Thus a hemolytic anemia is seen.[22] In addition, these cells sludge more readily intravascularly and are less able to carry oxygen to the tissue than a normal erythrocyte. In this case, areas of anoxia occur, the so-called "infarcts." However, no evidences of vascular occlusion, *i.e.*, thromboses, have been found originating from a defect within the erythrocyte.[22] Emboli to viscera from bone infarcts have been described.[13,21]

CLINICAL MANIFESTATIONS

Hemoglobin S-C disease and sickle cell anemia have many manifestations in common; however, there are differentiating factors, as shown in Table 1. A sickle cell

Table I

FACTORS DIFFERENTIATING HEMOGLOBIN S-C DISEASE AND SICKLE CELL ANEMIA

	Hemoglobin S-C+	Sickle Cell Anemia
Age at onset	26	Before 10
Average age	34	17
Hemoglobin (gm. %)	>11*	<9*
Appearance	Normal	Asthenic
Spleen	50% enlarged	Rarely palpated after age of 10
Vitreous humor hemorrhage[8]	+	Rare
Painless hematuria[2]	+	Rare
Crises during pregnancy[1]	+	Rare
Target cells	>50%	<30%
Sickled cells on peripheral smear	Rare	Common

* Asymptomatic.
+ Data from 99 cases (14 from the Temple University Hospital and 85 from the literature).

anemia patient with manifestations of hemoglobin S-C disease is rare, but the reverse is not true, excepting for the one characteristic of "appearance." Hemoglobin S-C disease can mimic sickle cell anemia.

ROENTGEN FINDINGS

Rose,[19] in 1929, reported the roentgenographic finding of a widened diploë in cases of sickle cell anemia. In 1937 Diggs *et al.*[6] presented a comprehensive study of the roentgenographic changes found in sickle cell anemia. Since that time many articles have been published amplifying these findings, but few authors have described the roentgen findings in hemoglobin S-C disease.[5,7,10,11,17,20,21,23,24]

The roentgen findings of 14 patients with S-C disease examined in this institution and of additional cases found in the literature are presented.

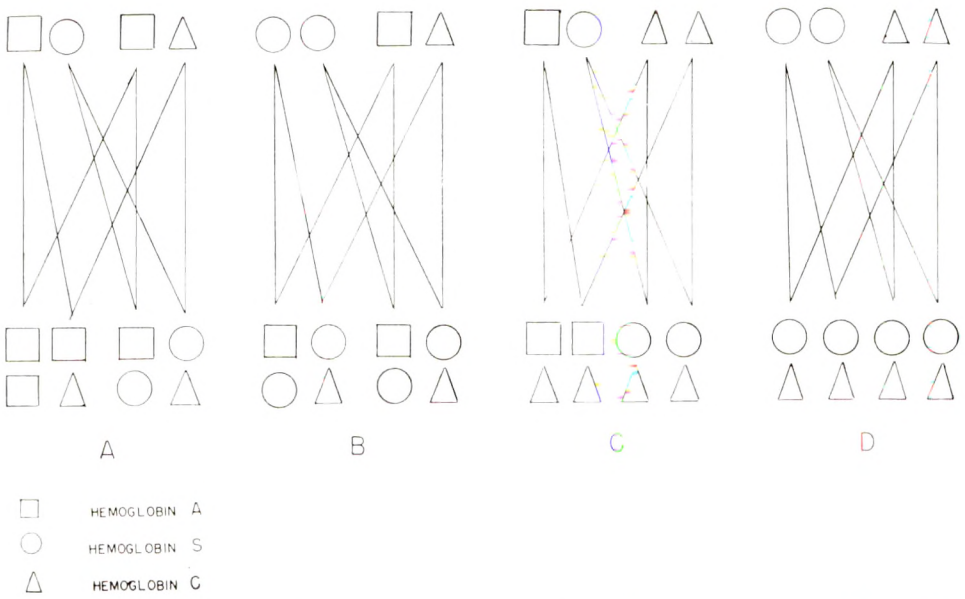


Fig. 2. Each parent has two genes for hemoglobin and, therefore, there are four possibilities for the genetic hemoglobin type of the offspring. Shown are four possible combinations that can produce at least one hemoglobin S-C genotype.

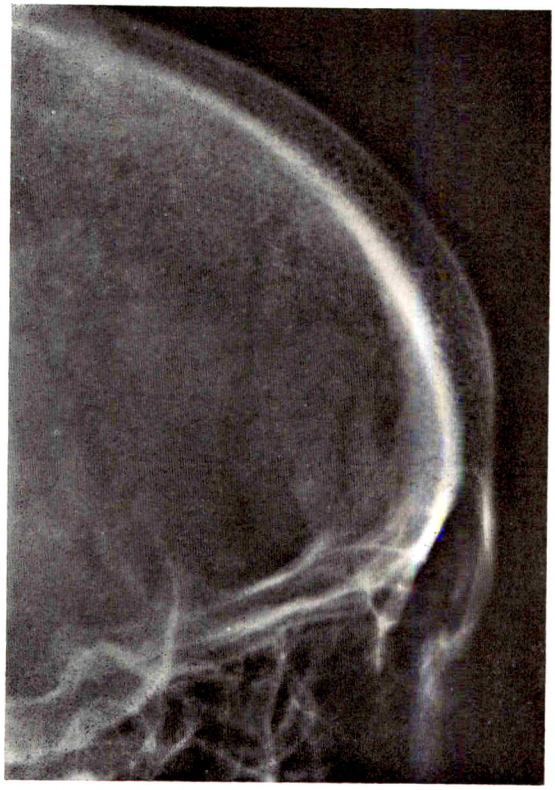


Fig. 3. A 19 year old female who had hemoglobin levels varying between 7.3 gm. per cent and 10.1 gm. per cent. She first presented with anemia at the age of 8 and had major episodes of postpartum hemorrhage with her pregnancies. The roentgenogram shows the classical widened diploë and the thinned outer table of the skull. There is a normal inner table.

A. osseous system

The osseous manifestations seen in hemoglobin S-C disease are the same as those described for sickle cell anemia, the sequelae being marrow hyperplasia and bone infarction. Secondary to anemia, there is a hyperplastic response of the bone marrow which produces a coarsened trabecular pattern (destruction of the finer trabeculae). There is also overtubulation of cylindrical bones with a narrowing of the cortex. Bone infarction occurs only in the erythropoietic and hemolytic anemias associated with hemoglobin S.

1. Skull. The classic findings of a widened diploë and a thin outer table of the skull, as shown in Figure 3, were encountered in only one out of the 9 cases which were examined. However, an incidence as high as 33 per cent has been reported.[5] The progression of the marrow hyperplasia producing the "hair-on-end" appearance was not found by us, nor is it reported in the literature.

The finding of localized radiolucencies in the diploë of the skull has been described in both sickle cell anemia[15] and hemoglobin S-C disease.[5] Although this has been referred to as "infarction," the roentgen appearance differs greatly from that of infarction in other bones and may instead represent localized marrow hyperplasia. Only one of our patients of 9 examined (Fig. 4) exhibited this finding (the same patient who presented the widened diploë). Only Cockshott,[5] in the review of the other hemoglobin S-C disease patients, mentions this finding.

2. Vertebral Column. Coarsening of the trabecular pattern of the vertebral bodies was the most common osseous finding in our cases, with an incidence of 33 per cent (Fig. 5). This agrees well with the incidence rate noted in the literature.[5,17,23] The progression to "fish vertebra," the sequel of disk invagination into the cancellous bone of the body, was noted in only 2 cases out of 12

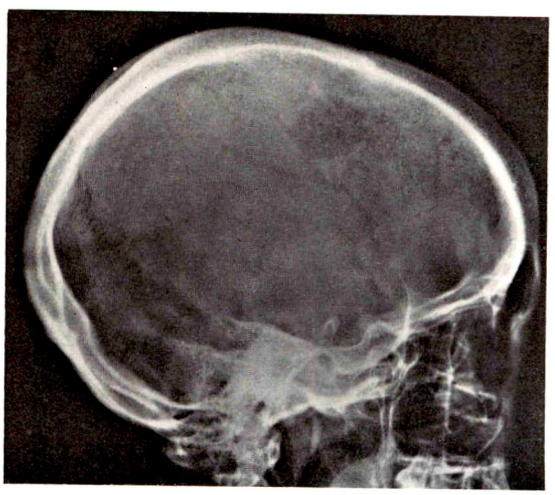


Fig. 4. Same patient as in Figure 3. The roentgenogram shows a radiolucent area in the diploë that is well marginated. This has been described as the residuum of an "infarct."

examined (Fig. 6). This occurred in the thoracolumbar region, as seen in sickle cell anemia. No evidence of collapse or infarction of the vertebral bodies has been noted in hemoglobin S-C disease.

3. Extremities. Overtubulation of the small bones with thinning of the cortex is mainly reported in children, since these bones are erythropoietic centers early in life.[15] This change, as noted before, is secondary to marrow hyperplasia and since we have not examined patients in the young age group, we did not encounter this manifestation.

Bone infarction is seen in 3 distinct patterns: (*a*) cortical; (*b*) medullary; and (*c*) epiphyseal (aseptic necrosis). Each type produces a characteristic roentgenographic pattern. The occurrence of cortical infarcts has been described in the literature,[5,17] but we have not seen an example of either the acute or chronic state. The roentgenographic identification of this manifestation is frequently missed since it takes ten to twenty days after the onset of acute symptoms (pain and swelling) for the periosteal proliferation to occur.[15] Frequently, roentgen examination is made at the time of the acute symptoms, with resultant normal roentgenograms. If no follow-up studies are obtained this lesion remains undetected. The healed state produces an area of localized cortical thickening.

The second type of infarction, medullary

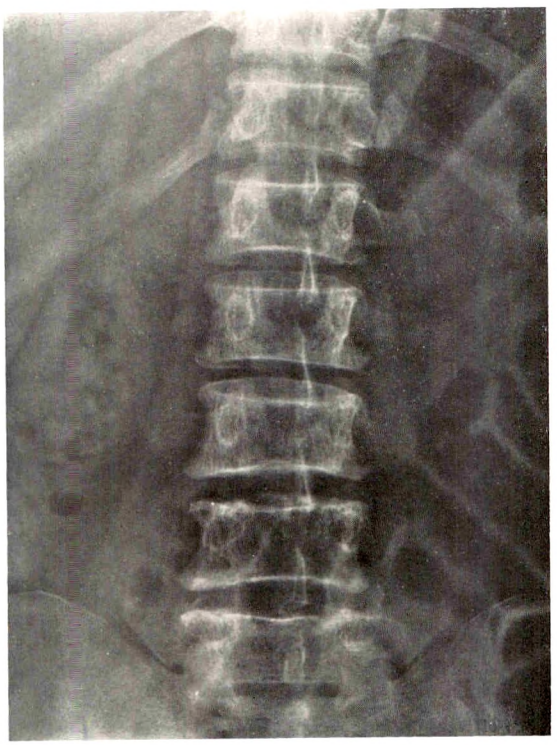


Fig. 5. A 48 year old female who had hemoglobin levels varying between 7.4 gm. per cent and 11.5 gm. per cent. She first presented with symptoms of abdominal pain (a common complaint) at the age of 31. She had bilateral femoral head aseptic necroses. Abdominal roentgenograms showed an enlarged spleen and an adynamic ileus. The above roentgenogram demonstrates a coarse trabecular pattern of the vertebral bodies but no "fish vertebra." This is the most frequent osseous roentgenographic finding.

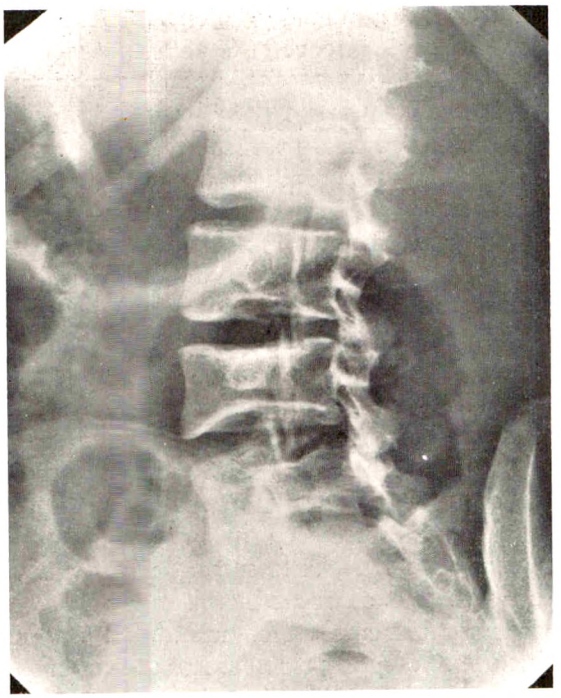


Fig. 6. A 29 year old male who had hemoglobin levels varying between 8.6 gm. per cent and 12.8 gm. per cent. He first presented with symptoms—arthralgia and back pain—at the age of 2½. He has had six hospitalizations for severe exacerbations of his symptoms. This patient had an episode of cardiac enlargement. The skeletal survey was within normal limits excepting for the coarsened trabecular pattern and "fish vertebra." The roentgenogram shows disk invagination into the bodies of L-2 to L-5. Of the cases we examined, this patient showed the most advanced spine changes.

infarction, is usually silent. Roentgenographically, this presents as an amorphous deposit of calcium within the marrow cavity, usually close to the distal ends of the bones. Since the process does not affect either the periosteum or the articular cartilage, the patient is commonly without symptoms. This manifestation has been reported in hemoglobin S-C disease,[5,7,17] but we have not observed it in our material.

The third type of infarction, aseptic necrosis, affects the very distal aspects of the long bones, specifically the articular surface. The most common site involved is the femoral head. This process, the most severe of all the types of infarction, is the one most often seen in hemoglobin S-C disease. In fact, it is second in frequency only to the coarsened trabecular pattern seen in the vertebral column. We found an incidence of 4 aseptic necroses of the hips in 3 patients out of 11 examined. Golding *et al.*[7] report an incidence of 70 per cent in the 19 patients they examined. No instances of aseptic necrosis of the humerus were found, although this is reported as the second most frequent site for aseptic necrosis. Smith and Conley[23] feel that the finding of an aseptic necrosis is almost indicative of a diagnosis of hemoglobin S-C disease in a patient demonstrating sickled cells; however, we have found aseptic necrosis in sickle cell anemia patients, as has Golding and his co-workers.[7]

The patient may present with symptoms months before any roentgenographic signs are noted, as shown in Figure 7 *A*, *B*, and *C*. As the process progresses, marked degeneration of a joint, especially in weight-bearing areas, is demonstrated.

A case of osteomyelitis in the extremities has been described in hemoglobin S-C disease by Sackner *et al.*;[20] however, the etiology was not *Salmonella*, as has been reported in sickle cell anemia. We have not seen a case of osteomyelitis in any of our patients.

B. VISCERA

Roentgenographically, the viscera also present some of the features of sickle cell anemia, but the similarity is less marked than that in the osseous system.

1. Lungs. Pneumonia, often noted in

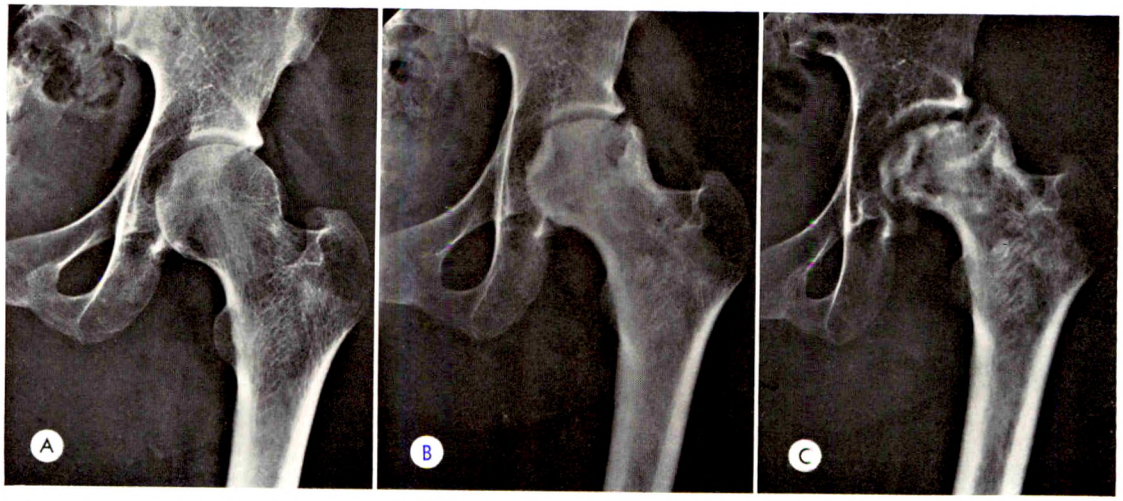


FIG. 7. A 33 year old female whose hemoglobin levels varied between 5.9 gm. per cent and 11.2 gm. per cent. She presented with anemia at the age of 21, her first symptom referable to hemoglobin S-C disease. The only osseous finding was aseptic necrosis of the left hip. The remainder of a skeletal survey was negative. Two episodes of "pneumonias" were noted. The patient presented with severe left hip pain in September, 1958. A roentgenogram (*A*) taken in October, 1958 is normal. The patient continued to complain of severe pain, but further studies were not ordered until November, 1959. At this time moderately advanced aseptic necrosis of the femoral head is seen (*B*). A repeat study in September, 1960 (*C*) shows a far-advanced aseptic necrosis with complete destruction of the articular surface. The patient continued to walk during the entire interval.

sickle cell anemia, was the most common roentgen finding in our cases of hemoglobin S-C disease, occurring in 45 per cent of the 11 patients examined. Others have also observed this but have not given an incidence rate.[11,23,24] The pneumonias are usually patchy in nature and the question arises as to whether the parenchymal densities do not, in fact, represent segmental pulmonary infarctions. Here, as in sickle cell anemia, the answer is not known; however, there have been instances of pulmonary infarction from marrow embolization following bone infarctions.[13,21]

The pneumonias seen are usually associated with crises and here also an unanswered question arises. Are the crises precipitated by the pneumonia or is pneumonia a manifestation of the crises?

2. Heart. Cardiac enlargement is rarely seen in hemoglobin S-C disease. One patient out of 10 in our group showed a transient episode of generalized cardiac enlargement. Hook and Cooper[10] and Smith and Conley[23] found cardiac enlargement to be infrequent. This is in contrast to sickle cell anemia where generalized cardiac enlargement is a frequent finding.[14]

3. Gallbladder. Cholelithiasis has not been described in hemoglobin S-C disease. Three patients in our series had cholecystograms and normal gallbladders were demonstrated. This is in contrast to cases of sickle cell anemia where the frequency of gallbladder stones is above average. This finding is to be expected since the hemoglobin S-C disease patients do not have as severe a hemolytic anemia as the true sickle cell anemia patients do and consequently have lower bilirubin levels. However, it is to be expected that the hemoglobin S-C disease patients will develop cholelithiasis, on the basis of their longevity, and that the underlying causes may not be calcium bilirubinate.

4. Spleen. Three out of the 12 patients examined in our series showed an enlarged spleen (Fig. 8). This is not a common finding in sickle cell anemia, as noted before. Also, in one of our cases an increased radiodensity of the spleen was observed (Fig. 9).

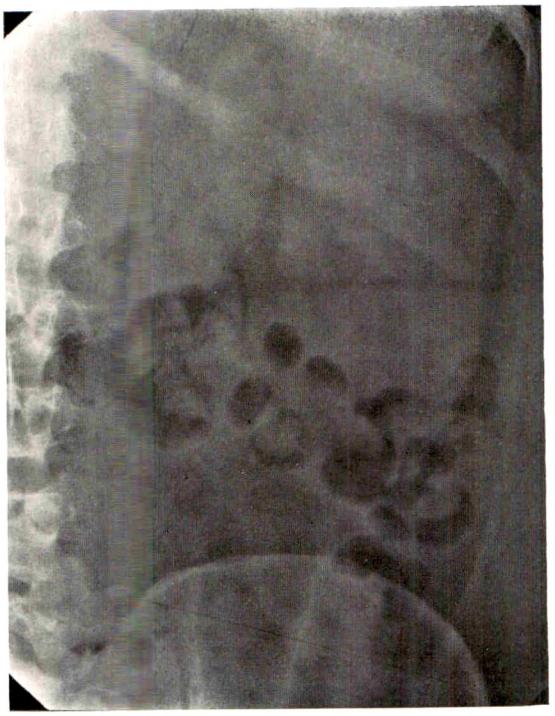


Fig. 8. A 20 year old female who was diagnosed as having sickle cell anemia at the age of 35, after one year's history of symptoms related to anemia. The hemoglobin values ranged between 8.2 gm. per cent and 8.6 gm. per cent.

The roentgenogram shows an enlarged spleen depressing the gas-filled splenic flexure of the colon.

This possibly is produced by microscopic calcification within the splenic pulp, which is known to follow splenic infarctions.[23] Hemosiderosis may be the etiology of the dense appearance of the spleen.

5. *Kidneys.* No urographic findings were demonstrated in hemoglobin S-C disease. The one patient in our series who had hematuria had normal intravenous urograms and retrograde pyelograms. Other investigators have also been unable to demonstrate renal lesions. In instances where nephrectomies have been performed to control bleeding, no cause of the bleeding could be found on microscopic examination.

DISCUSSION

Hemoglobin S-C disease has replaced sickle cell anemia as the hemoglobin S hemoglobinopathy capable of a wide range of symptoms. Patients can be affected with a

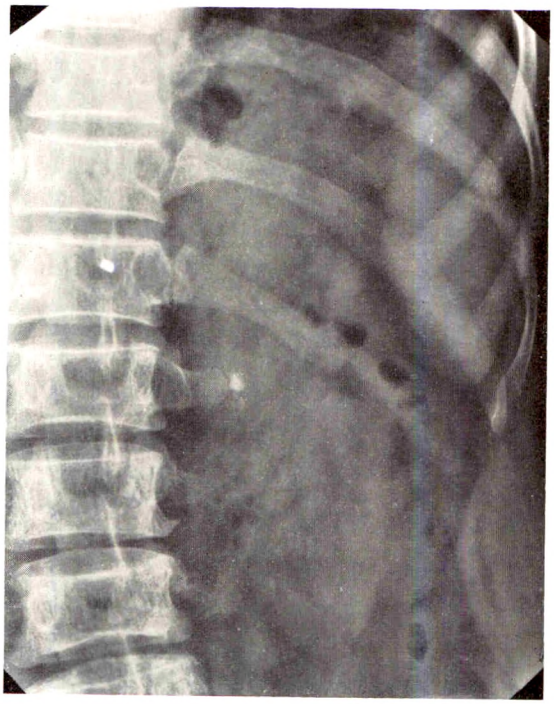


Fig. 9. Same patient as in Figure 3. The roentgenogram shows a spleen of increased radiodensity in comparison with the surrounding tissues. We feel that microscopically this may represent a calcified spleen. Another possibility is hemosiderosis of the spleen. No pathologic proof is available.

severe manifestation of this disease within the first year of life, or can live out an asymptomatic life. This is opposite to the present concept of sickle cell anemia, in which those affected manifest their disease before the age of ten and rarely live beyond their forties. Both disease entities have the same underlying pathologic process. As would be expected, hemoglobin S-C disease is generally the more benign process—falling between sickle cell trait and sickle cell anemia in degree of severity. Thus, both its clinical and roentgenologic findings are much less prominent than those in sickle cell anemia. In fact, 40 per cent of the patients examined by us showed no roentgenographic changes suggestive of hemoglobin S-C disease. This is felt to be extremely significant, since the population examined was a symptomatic one. It is probable that patients with no symptoms, *i.e.*, those with a more benign course, would present even fewer roentgenographic signs and that the findings of a roentgenographic survey of known hemoglobin S-C disease patients would prove less definitive than those described here.

Hemoglobin S-C disease cannot be specifically diagnosed roentgenologically; however, when roentgen changes similar but not as pronounced as those seen in sickle cell anemia are present and normal to low normal hemoglobin levels are found in a patient well into adulthood, hemoglobin S-C disease must be considered. A major differentiating roentgenographic finding in hemoglobin S-C disease is the presence of aseptic necrosis. Considering the generally benign manifestations of the disease, the frequently encountered aseptic necrosis is a striking feature, especially since this is one of the more severe signs of the hemoglobin S hemoglobinopathies. We feel that the possibility of hemoglobin S-C disease must be strongly suggested when aseptic necrosis is seen in a patient known to harbor sickle cells.

SUMMARY

1. A "new" disease, hemoglobin S-C disease, a variant of sickle cell anemia, is now a well known clinical entity.

2. The features that differentiate hemoglobin S-C disease from sickle cell anemia, both clinical and roentgenographic, are presented.

3. Hemoglobin S-C disease generally has a more benign course and less pronounced roentgenographic manifestations than sickle cell anemia.

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ADDENDUM

Since this article was submitted for publication, Drs. Widen and Cardon (*Ann. Int. Med.*,

1961, *54*, 510–521) have reported a case and a review of other possible cases of *Salmonella* osteomyelitis associated with heterozygous hemoglobin S hemoglobinopathies.

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ROENTGENOGRAPHIC AND CLINICAL APPRAISAL OF SICKLE CELL-HEMOGLOBIN C DISEASE*

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WITH the year 1961, sickle cell-hemoglobin C disease enters its second decade as a recognized pathologic entity. As one of the hereditary hemoglobinopathies, its existence is determined by genes which govern the structure of the hemoglobin molecule.[14] In addition to normal hemoglobin, called A, the genes for several alphabetically designated mutants exist. Our concern rests with the mutants S and C. When heterozygous with the gene for normal hemoglobin, no overt disease is present. However, when homozygous or when paired with another mutant gene, clinically perceptible disease *may* evolve, but expression of the genotype is inconstant.

For the most part, these diseases were defined as discrete entities in the laboratory within the past decade and are still dependent upon complicated laboratory procedures for their identification. The gross modalities of clinical medicine may serve to define the perimeters of these entities in a general way, but wide intragroup variation precludes precise diagnosis. The diseases may produce, in bone and viscera, certain morphologic changes which may be demonstrated roentgenographically. When present, these roentgenographic signs offer a means of diagnostic refinement; their absence in some individuals not only frustrates the diagnostician, but points up the enigma inherent in this pathophysiologic involvement of the erythron.

MATERIAL

Clinical and roentgenographic findings in a group of 25 patients with electrophoretically proven sickle cell-hemoglobin C disease were evaluated. The youngest patient was 3 years old, the oldest 61. Their inclusion in this series is the result of their having been the subject of roentgenologic consultation within the past 3 years in Parkland Memorial Hospital, Dallas, Texas. In all cases efforts were made to obtain a complete roentgenographic survey which usually consisted of 2 views of the chest, the spine in 2 projections, the abdomen which included the pelvis and the femoral heads, 2 views of the skull, at least one long bone and, in most cases, views of the mandible.

The medical records of each patient were reviewed and inadequacies in clinical laboratory data resolved when possible. Sickling of the red blood cells was observed in each of the patients. The presence of S and C hemoglobin was determined by means of paper electrophoresis and, in several of the patients, by starch block analysis. In most cases, hemoglobin and hematocrit determinations were obtained when the patient was not in crisis and was free of intercurrent disease. Findings on physical examination and past medical history, especially as they pertained to the common manifestations of the sickle state, were carefully codified. In compiling clinical data, efforts were made to obtain reliable information concerning the age of onset, frequency and severity of crisis.

In evaluating the nature of the roentgenographic findings as seen in this disease, material from all 25 patients was employed. The group of 25 patients, although constituting an estimated 14 per cent of the S-C genotype in the community, is not large enough to lend statistical validity to estimates of the true frequency of the signs

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observed. In attempting to assess correlation between clinical course and observed roentgen abnormality, the data from only 20 adult patients were chosen as being most comprehensive.

Discussion centers primarily on the patterns in the sickle cell-hemoglobin C group. However, brief comparison will be made with the findings as observed in a group of 56 patients with proven sickle cell anemia and with 3 patients with hemoglobin C disease. The clinical and roentgenographic data in these latter groups were assembled and evaluated as described above.

ROENTGENOGRAPHIC FINDINGS

In considering the roentgenographic signs, they might be categorized according to the methods of others:[11] (a) bony changes which result from hyperplasia of the erythropoietic elements; (b) bony changes which result from infarction; and (c) changes in the viscera.

Marrow Hyperplasia. The life span of the red blood corpuscle in S-C disease is appreciably less than normal. The patient suffers a chronic hemolytic anemia. In some individuals, intercurrent hemolytic crises compound the anemia. The resultant diminution in oxygen tension may be a stimulant to increased red blood cell formation. With constant stimulation, erythropoietic elements become hyperplastic. The marrow shares its milieu with cancellous bone in, for example, a vertebral centrum. Speaking teleologically, the marrow overcompensates and crowds out, perhaps by pressure, the smaller trabecular spicules thus diminishing the solid bony structure. Functionally, this weakens the bone. With weight bearing, changes in contour, the result of compression, may occur. Roentgenologically, the bone becomes more radiolucent and the trabecular pattern appears coarsened. The remaining trabeculae in such a bone stand out because they are thrown into greater contrast by marrow of lesser density, marrow devoid of finer trabeculae. Classically, the shape of the centrum conforms to the noncompressible nucleus pulposus with resultant biconcave contour or the so called "fish vertebra." This contour was recognized in 8 of the S-C group. Occasionally, there is simple flattening or wedging (Fig. 1, *A* and *B*).

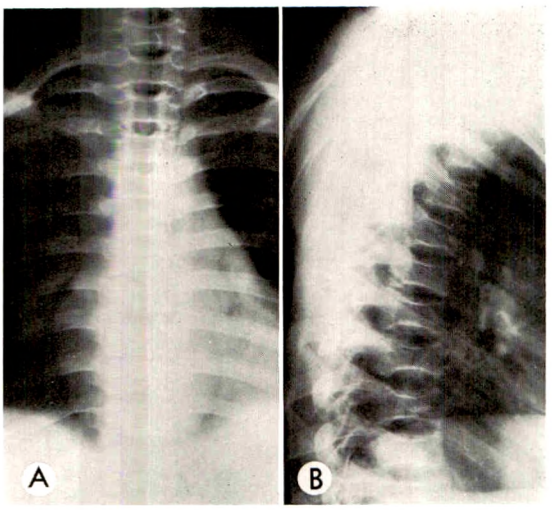


Fig. 1. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the thoracic spine of a 20 year old male with S-C disease. Vertebral centra show biconcave contour. Cancellous bone is radiolucent and the depressed end plates seem dense. The patient has suffered frequent crises for 15 years with pain in back and extremities. Hemoglobin S and C present in equal amounts. Hematocrit 32 per cent.

Similarly, hyperplastic changes may be reflected in bones which are not commonly the site of erythropoiesis such as the mandible.[15] Since the mineral content in the cancellous bone is diminished, body and ramus become more radiolucent. There is some thinning of the cortex. The stress of mastication dictates that bony strength be preserved and supporting trabeculae remain. They appear coarse and, in the alveolar bone between the roots of the teeth, a so-called "stepladder" pattern is detectable. The lamina dura is usually very prominent. These changes were present in 3 patients in the S-C group (Fig. 2, *A* and *B*).

Roentgenographic manifestations of marrow hyperplasia are said to be rare in S-C disease.[6] Our data would indicate that this is the most common abnormality. It is

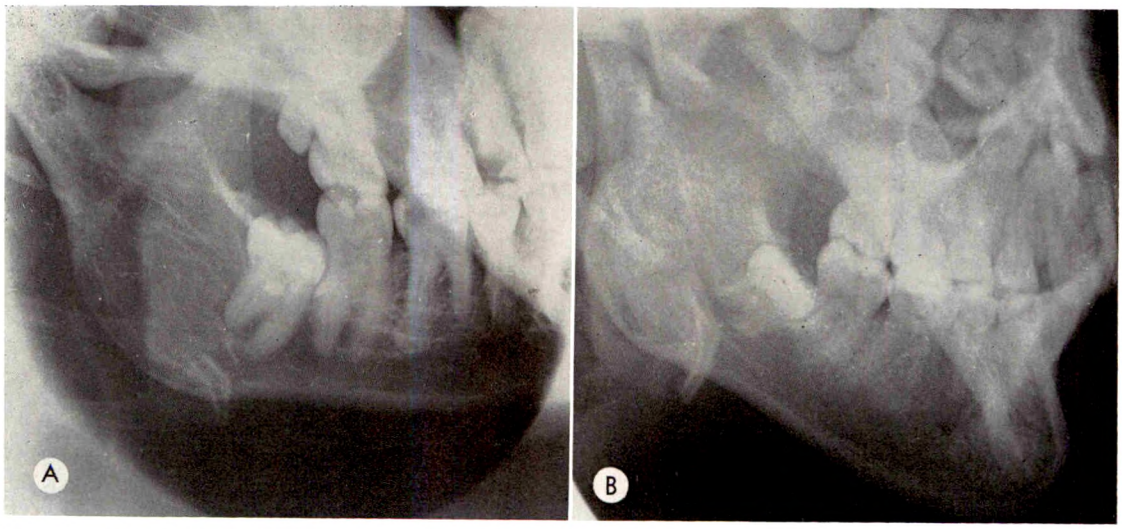


FIG. 2. (*A*) Mandible of a 22 year old patient with S-C disease. Note the increased radiolucency of the cancellous bone of the body and ramus. In the alveolar region, the remaining trabeculae are coarse and present a crude "stepladder" pattern between the roots. The lamina dura is usually more prominent. This was the only roentgenographic sign in this asymptomatic patient. (*B*) Roentgenogram of a normal subject for comparison.

observed in forms other than those just described in the spine and mandible. In the ribs and pelvis the appearance is that of increased radiolucency and coarse trabeculation. In the skull there is thickening of the calvarium, increased lucency or granularity and marked thinning of the outer table. These findings were present, singly or in combination, in 11 patients. They were marked in 8, minor in 3 and, though minor, were the only roentgenographic evidence of disease in 2 adults in whom hemoglobinopathy was clinically unsuspected. In the S-S group, hyperplastic changes were present, alone or in combination, in 70 per cent.

Bone Infarction. Besides marrow hyperplasia, another basic process, which may be responsible for the painful symptoms of disease, produces roentgenographic findings. This process, tissue infarction, is roentgenographically manifest in the S-C group. Infarction occurs as the result of vascular stasis and local anoxia. The basic mechanism is related to the molecular phenomenon which affects the configuration of the lines of force in the inner bindings of the peptides in the abnormal hemoglobin molecule. This aberrant linkage, under lowered oxygen tension, effects structural changes in the corpuscle to produce the sickled shape. With sickling, the viscosity of the blood increases, vascular stasis is implemented and the free flow in smaller vessels is impeded. Thrombosis and infarction may follow. The process is accompanied by pain. If a great enough volume of tissue is embarrassed, the result may be a roentgenographic finding.[12] In bone, the earliest sign might be the appearance of a zone of increased radiolucency. Later, with attempted repair, increased bony density is noted. Roentgen evidence of bone infarction is not uncommon in S-C disease. Sites of predilection include the long bones and pelvis and signs were present in 7 patients. Involvement of the humeral head and femoral head[16] was noted in our series. Often in epiphyses, the appearance is that of a mottled, chalky pattern which, after the acute episode, is not symptomatic. However, with the effects of weight bearing and the passage of time, deformity of the femoral head may ensue and there is a resultant debilitating arthritis (Fig. 3, *A* and *B*). Twenty per cent of the adults with S-C disease showed evidence of

aseptic necrosis of the femoral head with deformity. Marked changes of this type also occurred in the S-S group but were less common.

Occasionally, instead of producing an amorphous, chalky density within the medullary bone, the infarctive process results in a unique pattern which has been said to resemble a "bone within a bone."[12] This is not common but was observed in 5 adults with sickle cell anemia and in 3 of the S-C group (Fig. 4). The mechanism which produces this picture of a "cortex within a cortex" can only be surmised; it may be a stage in the narrowing of the marrow cavity noted in some older patients.[5] It is usually described as the result of localized anoxia subadjacent to the cortex with resultant infarction, the reparative process producing a zone of abnormally dense trabeculae. This pattern was seen only in adults. The time of onset may be related to the change from red to yellow marrow.



Fig. 4. "Bone within a bone." Forty-six year old patient with S-C disease, who suffered frequent crises with pain in extremities as a child. No symptoms were present in early adult life but the crises recurred in the third decade. Note the linear zone of coarse trabeculae which parallels the cortex resembling Paget's disease, but is unique in sickle states. The chalky density in the femoral neck and above the acetabulum on the left denotes other sites of bone infarct.

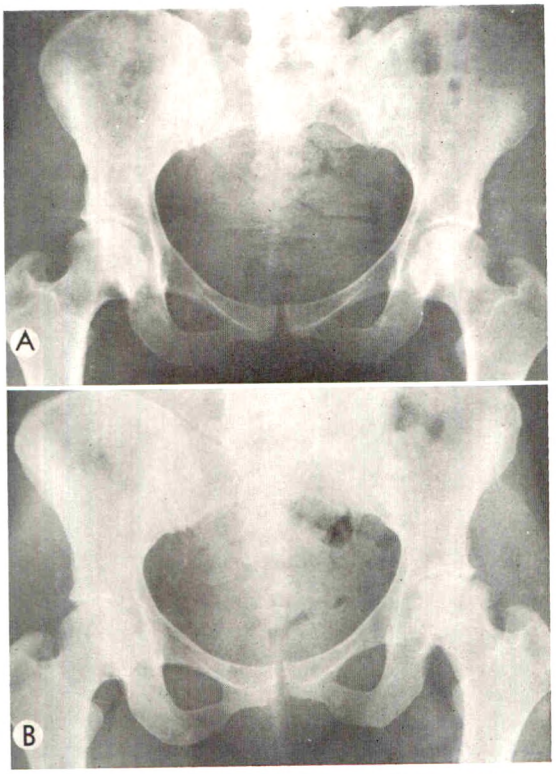


Fig. 3. Aseptic necrosis of the femoral head. Thirty-five year old patient with S-C disease. (*A*) Roentgenogram made when patient had experienced 2 weeks of pain in the left hip. No evidence of bony abnormality. The pain and limitation of motion increased. (*B*) Fifteen months later, there is fragmentation and deformity of the left femoral head. Note the mixed radiolucent and chalky appearance.

Visceral Changes. Of the changes which occur in the viscera in S-C disease, splenomegaly is most notable. In the younger age group, it may be primarily related to extramedullary hematopoiesis. This process, in some, may persist into adult life. However, splenic enlargement was usually observed as a transitory phenomenon accompanying crisis and the signs and symptoms of splenic infarct were usually present. This was roentgenographically demonstrated in 4 adults (Fig. 5).

Repeated episodes of splenic infarction are known to occur in S-C disease and may produce fibrosis and shrinkage. Splenic calcification has been described in S-S disease

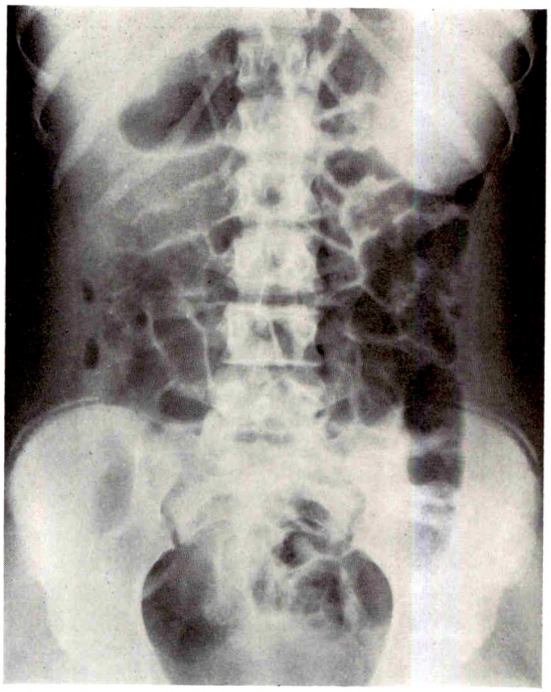


Fig. 5. Splenomegaly. Thirteen year old patient with S-C disease at the time of the first crisis which occurred following onset of pneumonia. One week later, the spleen was no longer palpable. Note the pattern of mild adynamic ileus which is often present during an "abdominal" crisis. There were no bony abnormalities.

as the end stage in this process.[10] It was observed in our series in 4 patients with the homozygous disease. The youngest of these was a 21 year old male. Since a similar pathologic process occurs in S-C disease, infarction, necrosis, hyalinization and eventual calcification might be expected occasionally in the heterozygous state. It is undoubtedly rare, but it was observed in one patient, a 46 year old male with S-C disease (Fig. 6). The density of this small splenic remnant is thought to be greater than that which might be produced by siderofibrotic tissue alone; calcium is probably present.

Gross hematuria, the result of ischemic changes in renal parenchyma, occurs in the sickle states usually in association with the trait or S-C disease.[3] Definitive diagnosis of this condition is not in the domain of radiology but information of value was obtained in one patient in whom inconstant filling defects, interpreted as blood clots, were demonstrated in the renal pelvis on pyelograms.

The morbid effects of infarction are nowhere more profound or life threatening than in the lung. Acute pulmonary infarction was observed only in the S-C group, and then, under circumstances which suggest a syndrome which has been described by others in the sickle state.[1,4,7,13,14] Three patients, all previously asymptomatic, suffered this complication during late pregnancy or in the puerperium in association with hemolytic crisis. Chest roentgenograms showed the infiltrative pattern in each of the 3 to be much the same. Initial roentgenograms, obtained very soon after the onset of pleuritic pain, showed only diaphragmatic elevation and accentu-

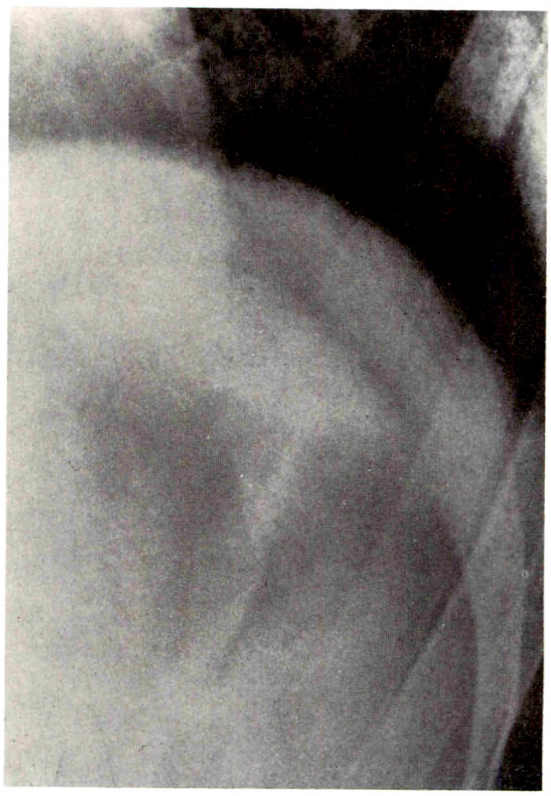


Fig. 6. Shrunken, calcified spleen. A 46 year old patient with S-C disease (see Fig. 4). This view of the left upper quadrant of the abdomen was taken from a posteroanterior roentgenogram of the chest. The small scarred spleen is faintly discernible. Its density suggests the presence of calcium.

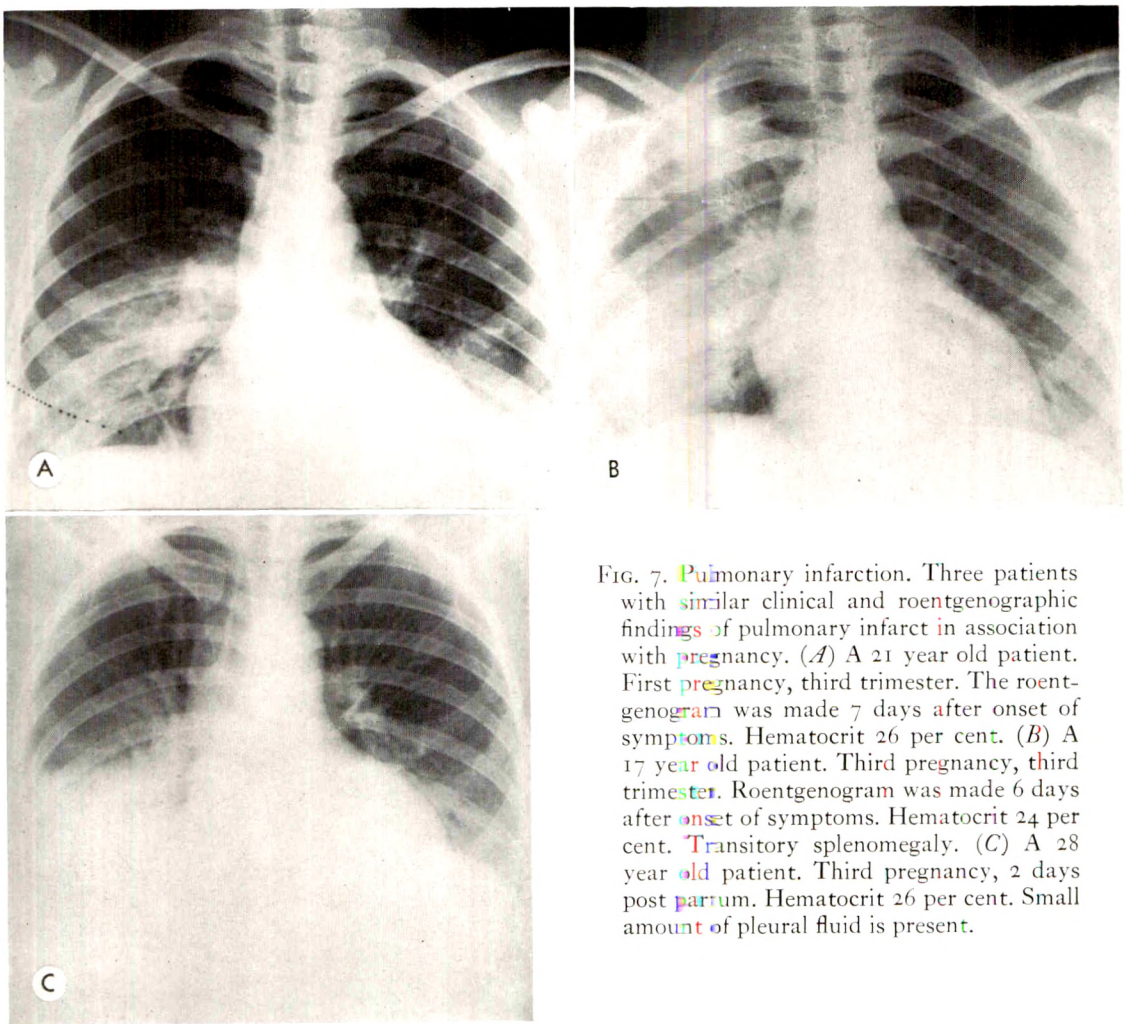


Fig. 7. Pulmonary infarction. Three patients with similar clinical and roentgenographic findings of pulmonary infarct in association with pregnancy. (*A*) A 21 year old patient. First pregnancy, third trimester. The roentgenogram was made 7 days after onset of symptoms. Hematocrit 26 per cent. (*B*) A 17 year old patient. Third pregnancy, third trimester. Roentgenogram was made 6 days after onset of symptoms. Hematocrit 24 per cent. Transitory splenomegaly. (*C*) A 28 year old patient. Third pregnancy, 2 days post partum. Hematocrit 26 per cent. Small amount of pleural fluid is present.

ation of basilar markings. Within 24 hours, however, dense confluent infiltration, usually basal, was seen. Bilateral changes may evolve. The distribution generally indicates involvement of more than one segment. A small amount of pleural fluid was demonstrated in one patient. Clearing occurred in 2 or 3 weeks, lagging several days behind the clinical improvement. Minor stranding persisted (Fig. 7, *A*, *B* and *C*).

Among the visceral abnormalities in true sickle cell anemia, cardiomegaly is by far the most common.[2] It was noted in 70 per cent of the S-S group and in 7 of these it was the only roentgenographic abnormality. Enlargement, when present, was slight to moderate and usually of a generalized type, without evidence of individual chamber predominance except in those few patients in whom the right heart, in response to chronic progressive thombotic involvement of pulmonary vessels or the fibrotic residue of repeated pneumonia, assumed the contour of cor pulmonale.

In sharp contrast to these findings in the S-S group, cardiomegaly did not appear as a roentgenographic manifestation of sickle cell-hemoglobin C disease. It was present in 3 patients in this group with hypertensive cardiovascular disease and the resultant left ventricular enlargement was felt to be unrelated to hemoglobinopathy. The presence of an enlarged heart in S-S disease and its absence in S-C disease is probably a

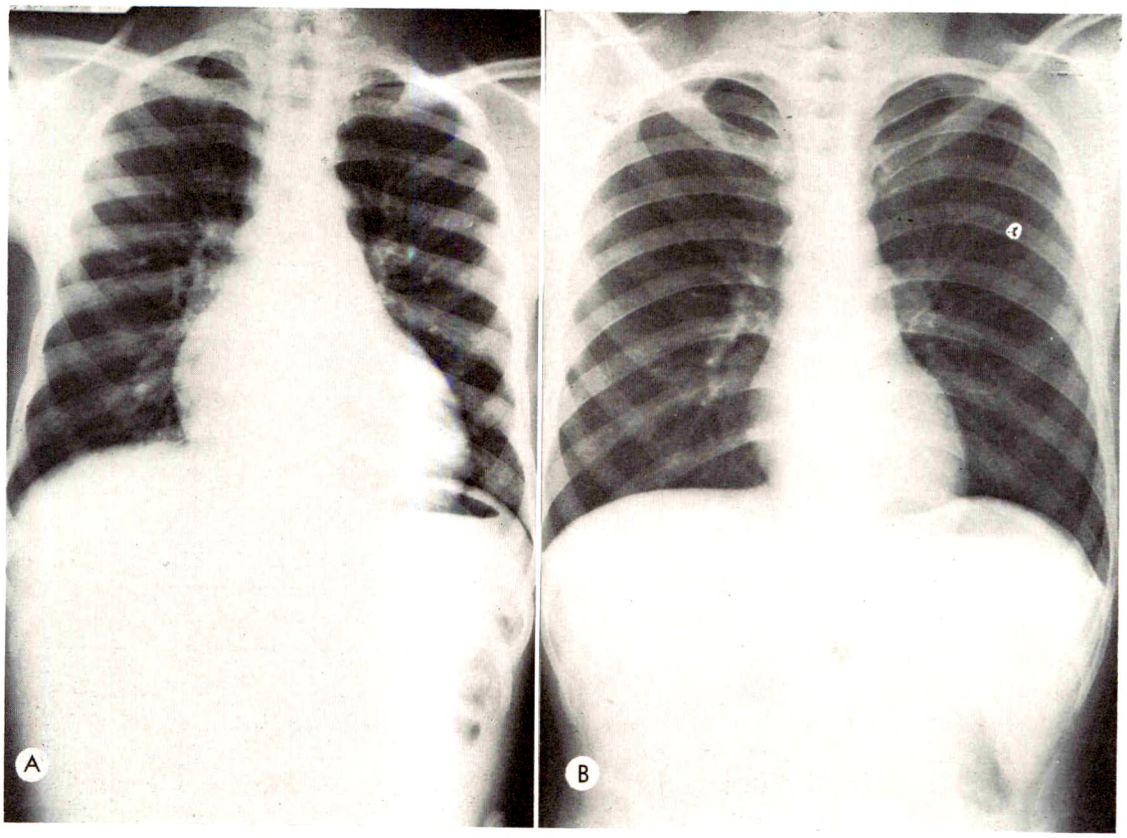


FIG. 8. Heart size in the sickle states. (*A*) A 22 year old patient with S-S disease. Hematocrit 27 per cent, hemoglobin 8.0 gm. Long history of frequent crises. A systolic murmur was present. The ribs show coarse trabeculae. The spleen (not shown) was small and calcified. Note cardiomegaly. (*B*) A 20 year old patient with S-C disease (See spine in Fig. 1). The heart is normal; the spleen was palpable.

function of the differing severity of the anemia in the two groups.[17] Hematocrit values above 30 per cent were the rule in those patients with S-C disease; the anemia was usually more profound in the S-S patient (Fig. 8, *A* and *B*).

In reviewing the roentgenographic findings in sickle cell-hemoglobin C disease, emphasis has been placed on the abnormalities. However, it must be stressed that about half the patients in this group had no roentgenographic sign of disease. The normal roentgenogram, therefore, is among the more common findings in the heterozygous state.

DISCUSSION

It is difficult to describe the clinical and roentgenographic findings in the "typical" patient with sickle cell-hemoglobin C disease. Within this group, sharing a common genetic fault, two differing clinical disease patterns are noted: the chronic, morbid disease replete with painful crises, and the relatively asymptomatic "laboratory" disease with absent or rare clinical manifestations. Those within each subgroup share a common clinical course. It is interesting, but perhaps not surprising, that the roentgenograms reflect the clinical course with some accuracy.

In considering each of the subgroups, the clinical history and complete roentgenographic findings in 20 adults with sickle cell-hemoglobin C disease were evaluated. There were 9 patients with the chronic, symptomatic disease. At the time of this study, they ranged in age from 18 to 59.

There were 11 patients without overt disease who had been relatively asymptomatic. Their ages ranged from 13 to 61. In considering the relatively asymptomatic subgroup, it must be acknowledged that the younger individuals, presently designated as "asymptomatic" may, in the future, suffer the morbid form of the disease. However, conversion to the overt disease is rare after the second decade and thus might be anticipated in only 2 of the 11. As regards the symptomatic patient, it is not implied that all so designated have equally severe disease. There is variation in type and degree of symptomatology, but all stand apart as clinically recognizable victims of the sickle state.

The clinical course in the symptomatic group was marked by recurrent, painful crises. The age of onset of crises was, in the majority, later than that usually noted in S-S disease. None were symptomatic in infancy but were first aware of their affliction in childhood or in the second decade. Once the pattern was established, however, painful episodes were often as frequent and severe as observed in the worst of the homozygous group. Typically, the patient in relating his past medical history described the course of a chronic disease which had profoundly affected his general health and well being. Occasionally, there was an asymptomatic interval, but recurrence of the chronic course was usual. Painful abdominal crises were noted but back and extremity pain was more common.

The classic complications of the sickle state were noted in this subgroup: recurrent pneumonia and pyelonephritis were common. There were instances of retinal detachment with vitreous hemorrhage, recurrent epistaxis, cholecystitis, gross hematuria and, as may happen in the abdominal crisis which mimics the acute abdomen, laparotomy for supposed surgically correctible disease.

It is of interest to note that, in the entire group with sickle cell-hemoglobin C disease, no evidence of active or healed leg ulcers was discovered. This stands in sharp contrast to the occurrence of this affliction in the group with sickle cell anemia where there was an incidence of 24 per cent.

In considering the roentgen findings in the symptomatic group, it is noted that each of the 9 had one or more major roentgenographic signs related to the skeleton. None showed splenomegaly, but one (Fig. 6) had splenic calcification. Bony changes resulting from marrow hyperplasia were as common as those due to infarction and 6 patients showed both. It appeared that the magnitude of the roentgenographic abnormalities increased correspondingly with the number of years of symptomatic disease. All of these 9 patients related a history of repeated painful crises over the past decade or more. In this respect, the age of onset of crisis seemed to play a role only insofar as it affected the duration of the clinical disease. However, early onset of crisis did appear to control the nature of spinal abnormality, *i.e.*, biconcave deformity was noted in those reporting morbidity in childhood; simple flattening was seen when the onset came later.[8]

In the other subgroup in which the expression of the disease was clinically benign, there were 5 patients in whom its presence was totally unsuspected and in whom the incidental demonstration of sickled red blood cells prompted electrophoretic evaluation of hemoglobin. They had no history of crisis nor of any of the commonly accepted primary complications of sickle cell disease. The remaining 6 patients, however, during an isolated episode, suffered clinical expression of their disease. Although not repeated, these episodes required hospitalization. In one, there was a severe hemolytic crisis accompanying pneumonia; in another, gross hematuria which responded to decapsulation of the affected kidney. The others were observed to have symptoms of the sickle state expressed as a complication of pregnancy. One of these patients suffered cardiac decompensation in the third trimester which responded to transfusion but not to digitalis. None of these had suffered a repetition and all but

one have been followed for over a year since the episode.

Where clinical disease lies latent, seldom or never expressed, roentgenographic evidence of the disease is rare and, if present, often transitory. Bony changes were seen only in 2 cases and in each instance occurred as an expression of hyperplasia of the marrow elements, limited to the mandible in one and to the skull in another. Splenomegaly was noted as a transient phenomenon in 3 patients, associated with solitary symptomatic episodes. None, in this group, showed evidence of bony infarction. The roentgenographic findings of pulmonary infarction have been described. It is interesting that it occurred, in the acute, fulminant form, only in the previously asymptomatic patient.

In comparing the clinical course and roentgenographic findings in the S-C group, it is apparent that there is a strong positive correlation between the severity of the past clinical course and magnitude and number of roentgenographic changes in the individual patient. This parallel is even more striking when roentgen evidence of bony infarction is employed as a measure of past morbidity. If the roentgenogram is of any prognostic value, it is apparently of a negative sort in that those with normal roentgenographic findings, although not heir to chronic repeated painful crises, are subject to the more catastrophic complications of the hemoglobinopathy.

Prominent among these more serious complications is pulmonary infarction. Typically, the episode is heralded by the onset of bone pain accompanied by increased anemia. This is followed by pleuritic pain and dyspnea. There is cough and sometimes blood streaked sputum. A febrile course may supervene. The auscultatory findings of consolidation are noted. The spleen may be palpable. There is tachycardia and accentuation of the second pulmonic sound. Electrocardiographic evidence of acute cor pulmonale has been observed. Homans' sign is not present. Sputum and blood cultures are sterile. Following depression of the hematocrit, there is an increase in reticulocytes and serum bilirubin. All of our patients recovered; clinical improvement was noted in 8 to 14 days. There was one fetal death.

Diagnosis rests primarily on clinical findings; however, chest roentgenograms of our 3 patients showed a basic similarity which, with each case, became more helpful in confirming the clinical impression.

It is generally accepted that the consolidation seen on roentgenograms represents the pulmonary infarction but the mechanism is debated; the process may be of embolic origin, the result of bone infarct. The bone pain which precedes pulmonary symptoms in some patients probably denotes infarction within the marrow. Small particles of necrotic marrow, having gained access to a vein, have been described in pulmonary vessels at autopsy. If large or numerous, such emboli could produce the pattern we have seen; but, extensive, confluent infiltrates are not the rule in fat embolus after fracture, and antecedent bone pain was not universal in our patients. It has been proposed by others that the process in the lung is not dependent upon embolization but upon local vascular stasis. Perfusion may be inhibited in the narrowed arterioles of hypoventilated lungs with resultant decrease in flow rate through the capillary bed. With stasis may come a diminution in oxygen tension to produce a favorable environment for the sickling of red blood cells leading to increased viscosity thus augmenting stagnation. Vascular stasis may produce ischemia with resultant infarction without major thrombosis.

There are no proven means to explain the higher incidence of this pulmonary catastrophe in pregnancy or its tendency to occur (in our experience) only in the sickle cell variant. There is an increase in erythropoiesis late in the third trimester of pregnancy. Is there a corollary between this fact and the supposition that larger areas of necrosis occur in the variants which have larger red blood cell masses? The answer may come from investigation in this direction.

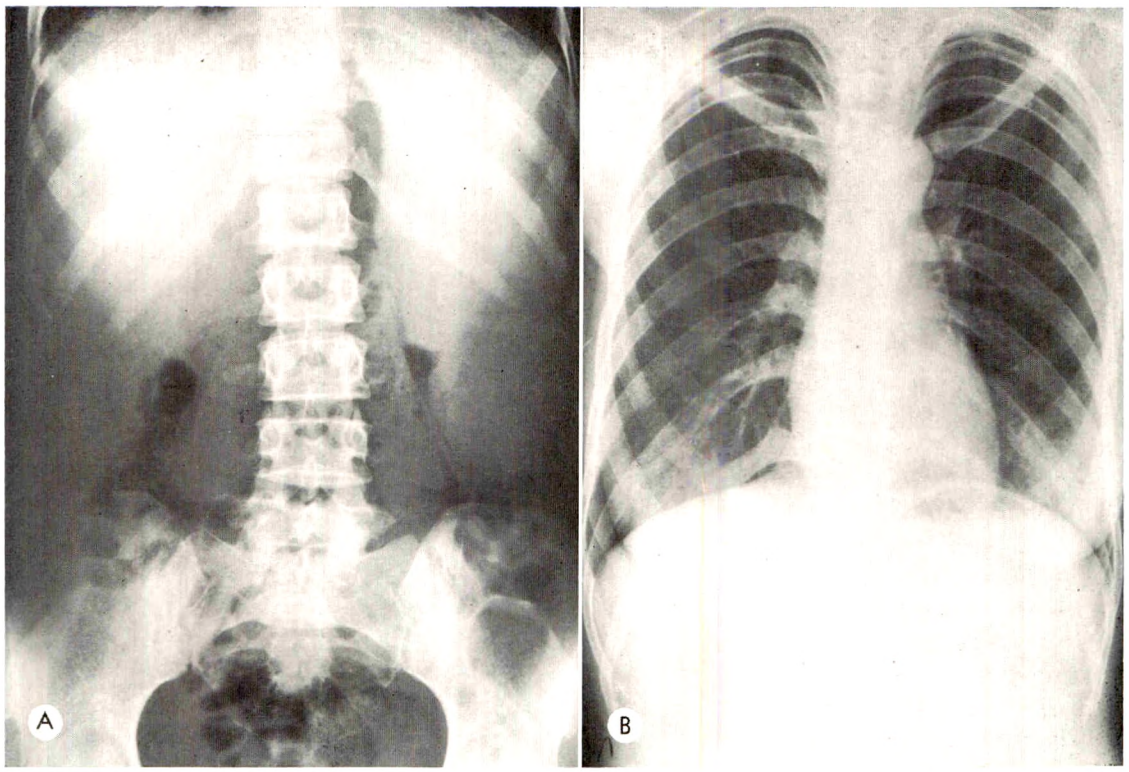


Fig. 9. (*A*) Abdomen and (*B*) chest of a 27 year old female with homozygous hemoglobin C disease. The massive spleen extends to the iliac crest. There were symptoms of "a full feeling in the stomach." At splenectomy, radiolucent biliary calculi were found. There are no skeletal abnormalities and no cardiomegaly.

Splenomegaly was found to be not uncommon in the adult in S-C disease. In the larger series of patients with S-S disease, roentgenographic demonstration of a large spleen was observed in only 3 patients, none older than 12 years.[9] It has been noted in adult patients with sickle cell anemia by others. Splenomegaly in the adult is quite common in another of the hereditary hemoglobinopathies. When the mutant gene C occurs in the homozygous state, hemoglobin C disease results.[18] It is a mild disease characterized by chronic anemia with occasional, episodic worsening of the anemic state. Splenomegaly, often marked, is noted as a manifestation of extramedullary hematopoiesis. Hematologically, many target cells are seen on smear and the life span of the red blood cell is reduced slightly. Three such patients were evaluated clinically and roentgenographically. All were in general good health. Only one complained of symptoms related to the disease: epigastric fullness of three years' duration (Fig. 9, *A* and *B*). This patient underwent splenectomy and, during surgery, biliary calculi were palpated. An increased incidence of gallstones might be expected in C-C disease as in other chronic hemolytic states. There were no osseous abnormalities. No cardiomegaly was present.

In seeking to distinguish S-S from its variant by roentgenographic means, we must concede that there is no pathognomonic sign. Indeed, the endeavor would seem hopeless when one considers that (a) identical roentgen abnormalities are found in individuals of each group, (b) within each group there are individuals who, roentgenographically, are indistinguishable from the normal. However, when the bony changes are present to indicate the existence of the sickle gene, we were encouraged to rely on the absence of cardiomegaly as a presumptive sign that the patient was of the S-C genotype.

SUMMARY

The clinical and roentgenographic findings in a group of 25 patients with sickle cell-hemoglobin C disease are described. The roentgen signs are discussed as they occur in the sickle cell variant, and brief comparison is made with a larger group of 56 patients with sickle cell anemia and a smaller group with hemoglobin C disease. Discussion centers on the differing clinical patterns within the S-C group: the chronic, recurrently painful disease; and the relatively asymptomatic process. These patterns are reflected on the roentgenogram. Pulmonary infarction was a prominent complication in 3 patients. The observations relating to pulmonary infarction, as it occurred in these patients, are discussed. Radiology would seem to be of no diagnostic value in the assessment of nearly half of the S-C group who have no roentgenographic abnormalities. Little encouragement is found in attempts to differentiate the heterozygous from the homozygous type by roentgenographic methods. The general absence of cardiomegaly in the S-C group may prove to be of some usefulness in this respect.

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BONE CHANGES IN HEMOGLOBIN SC DISEASE

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IN RECENT years there has been widespread interest in the abnormal hemoglobins by scientists and clinicians in many different fields of activity. Considerable light has been shed on some of these entities following research by biochemists, geneticists, hematologists, anthropologists, pathologists, physicians, surgeons and radiologists. In the population of Ibadan, in Nigeria, there is unfortunately a high incidence of several of these disorders which has afforded us in the radiology department an opportunity of studying many of these cases. This paper concerns itself with the roentgenographic manifestations of one of these disorders: hemoglobin SC disease, in a group of 117 persons.

In a previous communication[2] the features of this condition as observed in Africa were described in 20 patients; this report amplifies those findings in the light of our further experience. The roentgenographic findings are of importance as they can suggest the presence of the underlying disease to the radiologist before the diagnosis has been considered by the clinician.

Among the Negro races sickle hemoglobin (S) is the most frequently encountered abnormal hemoglobin. It may be present in a homozygous form as in sickle cell anemia (SS); in a heterozygous carrier state (AS) in combination with normal adult hemoglobin A; or lastly it may be associated with another abnormal hemoglobin such as hemoglobin C to cause (SC) disease. In Africa the S gene probably developed in at least 2 foci, one on the East and one on the West side of the Continent. Hemoglobin C appears to have originated as a mutation centuries ago in Ashanti. These abnormal hemoglobins have persisted because of balanced polymorphism and have spread over the years from their original areas due to intermarriage of different tribal groups consequent on wars, trade, migrations and slavery. Therefore individuals with hemoglobin SC are to be encountered particularly in Western Nigeria, Dahomey, Ghana, the Ivory Coast, the West Indies, Brazil and in the Southern States of America. In Ibadan 0.2 per cent of the population possess the SC gene.[18]

The principal clinical features of the disorder are a low grade hemolytic anemia and episodes of abdominal, joint and bone pains. A mild degree of anemia is frequent, due to the diminished survival time of the erythrocytes in SC disease as compared with normal controls. In Africa the anemia is often greater than appears to be the case in the New World because of the additional parasitic and nutritional insults which some of these patients face. The anemia is rarely severe unless acute precipitating factors such as pregnancy or persistent hematuria coexist.

The blood findings are characteristic and confirm the diagnosis. The peripheral blood film will reveal large numbers of target cells, polychromasia, possibly scanty reticulocytosis and a few sickled cells. The red blood cells show increased resistance to hypotonic saline. Fetal hemoglobin is raised. The diagnosis is finally established by paper or starch electrophoresis that will show the two bands for hemoglobin S and C.

Episodes of bone and abdominal pain are presumed to be due to reaction around infarcted areas. Sickling occurs due to a physical change in the hemoglobin in the cell when the oxygen tension becomes reduced. Because of their abnormal shape, such sickle cells tend to conglomerate

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together and clump, blocking small capillaries and so further impairing the oxygenation of the part and setting up a vicious circle resulting in vascular spasm, thrombosis and infarction.

The process of localized infarction is responsible for many of the clinical and roentgenographic features of the disease. There are, however, certain complications that occur in this condition such as unilateral renal hematuria, splenic abscess, and sudden death in the late stages of pregnancy or puerperium that are less well known and of obscure etiology.

METHOD

For the purposes of this survey 117 persons were examined. They can be divided into 2 main groups. First, 64 patients who came to the hospital with a complaint that on investigation was found to be related or caused by the presence of hemoglobin S and C. This clinical group consisted of 31 females and 33 males whose ages ranged from two to fifty-eight years. The second group consisted of those patients who were discovered to be possessors of the SC gene following routine electrophoretic investigation of their blood; these comprised 46 patients attending the antenatal clinic and 7 blood donors. All members of this second group were in their late teens or young adults and the large majority were young women. Consequently, this group cannot be considered to provide an entirely representative picture of the patient with this disorder. Nevertheless, at times, it is of interest to compare the "clinical group" with the second category of patients whose disorder was to an extent an incidental finding. Both groups of patients were submitted to a limited roentgenographic survey which varied in extent but included the following roentgenograms: lateral skull, anteroposterior projection of both hands and wrists, pelvis to include hip joints, and chest including shoulders. In addition, further roentgenograms such as those of the spine and long bones were taken when indicated by clinical findings. In the clinical group other areas were investigated if indicated. The antenatal group of patients were all examined after delivery.

Radiographically, there was no bone abnormality in 41 per cent of the antenatal patients, 13 per cent of the blood donors and in 9 per cent of the clinical group. All the remaining patients showed various abnormalities which can be divided for ease of consideration into bone changes following marrow hyperplasia, joint manifestations and lastly diaphyseal changes due to infarction with or without infection.

CHANGES CONSEQUENT ON MARROW HYPERPLASIA

The hyperplastic bone marrow causes thinning of trabeculae which in turn leads to weak osteoporotic bones that may become deformed. This process occurs to a much greater degree in homozygous sickle cell anemia and thalassemia as these are the conditions in which the hemolytic process is more severe. It appears that this type of change in SC disease is unusual elsewhere than in Africa. Recently, Golding *et al.*[5] did not observe any of these hyperplastic manifestations in their 19 patients with SC disease studied in the West Indies. However, Moseley[10] records spine changes due to hypertrophic marrow in patients observed in New York.

In Ibadan, we have noted occasional examples of definite thickening of the diplöe of the skull, significant osteoporotic changes in the ribs and spine and occasional manifestations of marrow hyperplasia in the hands and large phalangeal vascular foramina as previously described.[2] The alterations in trabecular architecture are, as a rule, most readily detected in the spine and pelvis and ribs. Essentially, the hyperplastic marrow causing thinning of trabeculae and disappearance of some trabeculae results in a "coarsening" of the bone structure. In the spinal bodies the transverse trabeculae disappear to a greater extent than the vertical supporting structure of the vertebral body. Despite this selective protective osteoporosis, some flattening of

bodies with biconvex intervertebral disks is common and fully developed "cod-fish" vertebrae are not rare (Fig. 1).

Diploic skull changes are infrequent and we have not observed the development of the "hair on end" appearance that is noted in some cases of homozygous sickle cell anemia.

Among the antenatal patients, one quarter showed one or more of the changes enumerated above but in only one of these was the change marked. Five of the 7 blood donors had generalized roentgenographic evidence of marrow hyperplasia and in one of these patients it was severe, with marked osteoporotic changes in the dorsal spine and "cod-fish" vertebrae. The numbers in this group are small and the high incidence of bone changes may be fortuitous. All in this group had a satisfactory hemoglobin level above 85 per cent, as otherwise they would not have been blood donors. It can be suggested that the hemoglobin concentration was satisfactory because of a particularly active marrow and hence the bone changes, but the numbers are too few for analysis. In the clinical group 45 per cent of cases showed some degree of hyperplastic bone changes and in 10 this was sufficiently marked for the spinal bodies to be obviously flattened.

Unusual alterations of rib trabecular structure have been the means of suggesting the diagnosis from a chest roentgenogram on several occasions (Fig. 2, *A* and *B*).

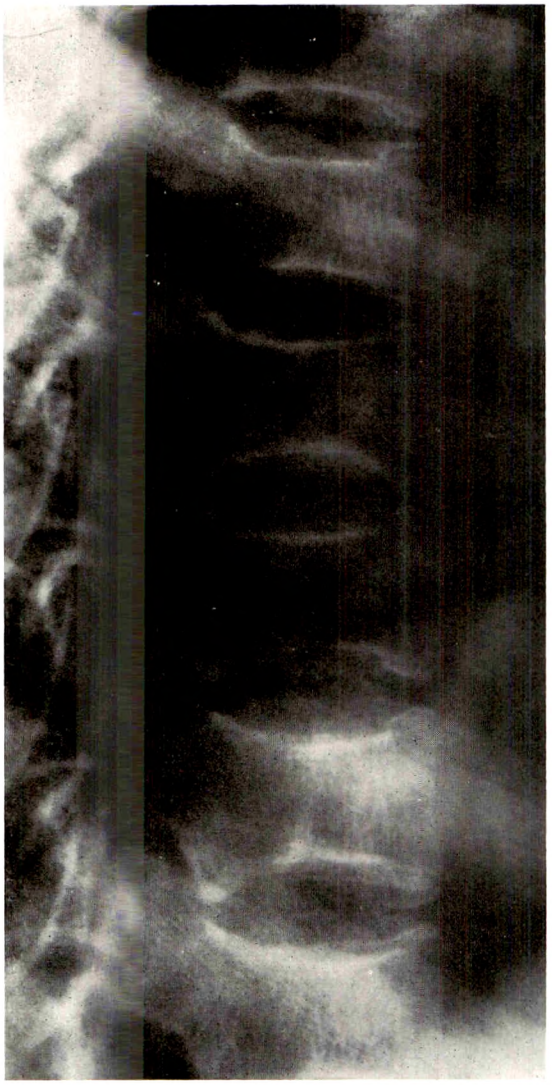


Fig. 1. Osteoporotic vertebrae with bulging intervertebral disks.

JOINT CHANGES

The flow of blood tends to be sluggish in the vascular loops that supply the cartilage at the ends of long bones and consequently the oxygen tension of the blood in these vascular channels can become reduced to the level at which sickling occurs. Blockage of capillaries, thrombosis, vascular spasm and infarction can readily follow. If the affected area is large enough, bone destruction ensues with later collapse of subchondral bone and distortion of the articular surface. The osteoblastic reparative process leads to areas of sclerosis. The development of these lesions has been well described and illustrated by Sherman[13] and Bobechko and Harris.[1] The hip joints are the most often involved and next in order of frequency are the shoulders, but other joints can be implicated. In our previous paper we stated that at that time we had not seen typical sickle hip changes in patients with homozygous sickle cell anemia. Since that time we have seen 6 examples in SS patients. In Nigeria, hemoglobin SC disease is responsible for hip changes far more often than is the homozygous disease. The high infantile mortality in true sickle cell anemia in

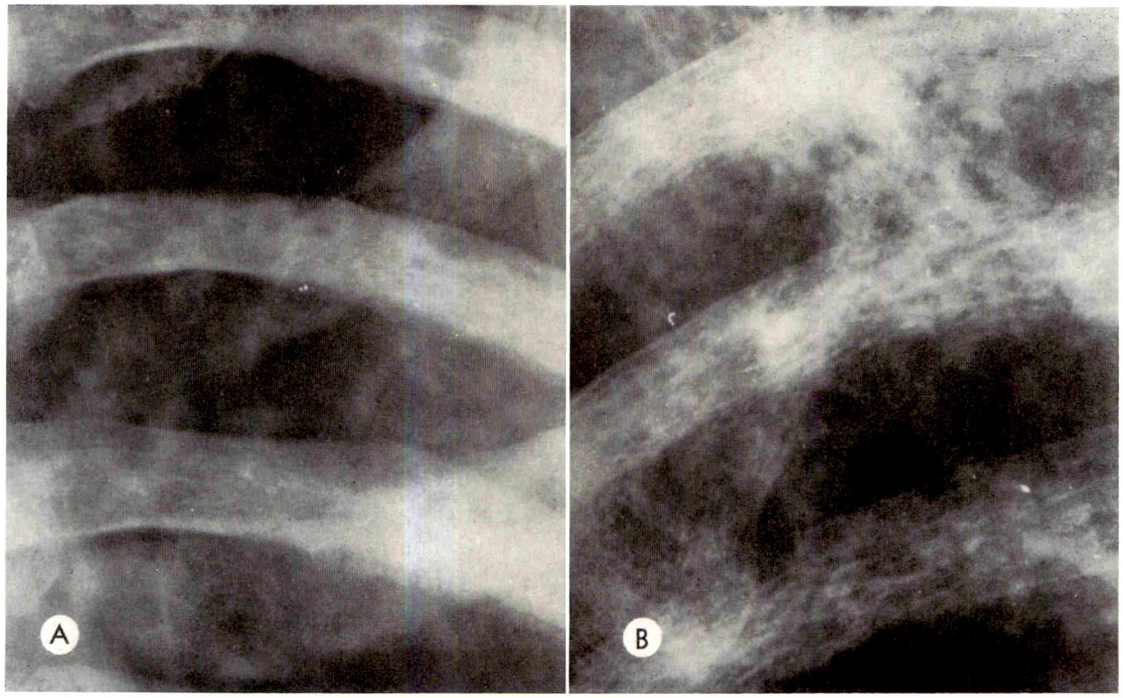


FIG. 2. (*A*) Unusual patchy rarefactions and areas of sclerosis in ribs. This has been seen in 2 cases of SC disease. (*B*) More common coarsening of trabecular pattern due to marrow hyperplasia.

Africa means that many children do not live long enough to develop hip disease.

The changes in the hip joints present at a later age than in Perthes' disease, and the two types of avascular hip changes can usually be differentiated by roentgenography. It has been stated by certain authors that Perthes' disease is exceptionally rare in the Negro,[14,15,16] whereas others find that the condition is by no means unknown.[3,7,11] It may be that Perthes' disease is very rare in Negroes of so-called Bantu derivation and more common among Negroes of West African ancestry.

As has been mentioned, the hip changes in SC disease occur at a later age than those in Perthes' disease and so the epiphyseal line is, as a rule, fused with the shaft, or is in an advance state of maturation when the patient is first seen. The avascular process is often focal or multifocal in SC disease (Fig. 3) rather than affecting the whole of the capital epiphysis as in Perthes' disease. Three types of roentgenographic changes that may be noted in the hips in this disease are: (1) coarse osteoporotic trabeculation and a degree of coxa vara; (2) a focal area of hip disintegration due to involvement of a sector of the femoral head consequent on vascular thrombosis. This is really a form of osteochondritis dissecans. As a rule, a fragment of the femoral, articular surface will be found to have become depressed following the collapse of the underlying, subchondral bone; and (3) generalized narrowing of joint space due to multiple, avascular focal lesions. These patients are usually older than those in the preceding groups.

Hip changes of the avascular type were observed in 12 per cent of the antenatal group and in 31 per cent of the clinical group. Among the latter were 10 males and 10 females whose ages ranged from ten to fifty-two years. The right hip was affected in 9 cases, the left in 11 and both hips in 4 patients.

Four patients had shoulder lesions of a type similar to those occurring in the hip.



Fig. 3. Series of roentgenograms showing several types of hip changes in SC disease.

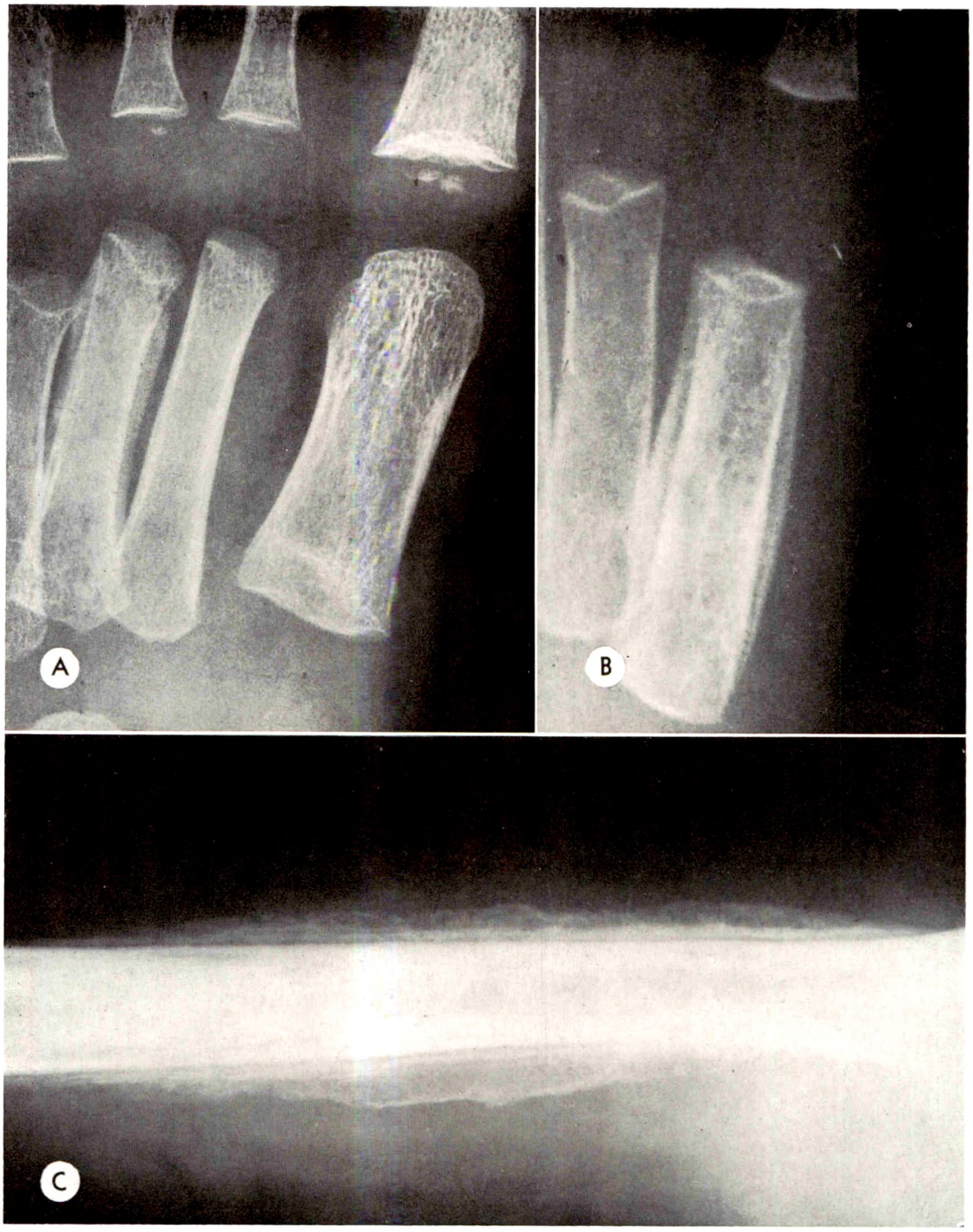


FIG. 4. Periostitis following diaphyseal infarction. The roentgenograms show this process in (*A*) a metatarsal bone, (*B*) a metacarpal bone and (*C*) a femur.

BONE INFARCTS AND INFECTION

It is frequently difficult to separate by purely roentgenographic criteria bone infarcts from bone infection, but a study of the clinical data combined with the examination of serial roentgenograms usually enables one to distinguish the two conditions. The difficulty arises because most episodes of bone infection follow an infarction. Since the organism causing the infection is frequently of a low grade, such as *Salmonella*, it is not surprising that the appearance is not specific and can be confusing. Golding[6] has claimed that bone infection occurs several hundred times more frequently in individuals with the S gene than in the normal AA genotype population.

The infarct most frequently involves the diaphysis but is visualized roentgenographically only if sufficient volume of bone is affected. Diaphyseal infarction occurs as a rule in children and those in their teens. Small infarcts may cause localized areas of bone destruction which are rapidly repaired. When significant segments of the diaphysis are involved, the features and appearances are characteristic. A thin line of periosteal new bone envelops the affected part of the diaphysis about a fortnight following the clinical episode of bone pain (Fig. 4, *A*, *B* and *C*). Middlemiss[9] has provided a good explanation of this process in pathologic terms. He suggests that following the death of bone the repair consequent on thrombosis of the nutrient artery involves particularly the small penetrating periosteal vessels. The periosteum is elevated and because of its osteoblastic activities lays down investing new bone similar to an involucrum. Gradually, this merges with the original bone and, as the process of repair continues, remoulding of the exuberant new bone takes place so that eventually the bone is restored almost to normal.

When the infarction affects the epiphyseal cartilage plate, various growth disturbances may follow, causing shortening of the bone or a deformed epiphysis. Damage to the epiphysis occurs most frequently in the hands. Several weeks after an episode of infarction, the epiphysis of a metacarpal or a phalanx of a child may be noted to be

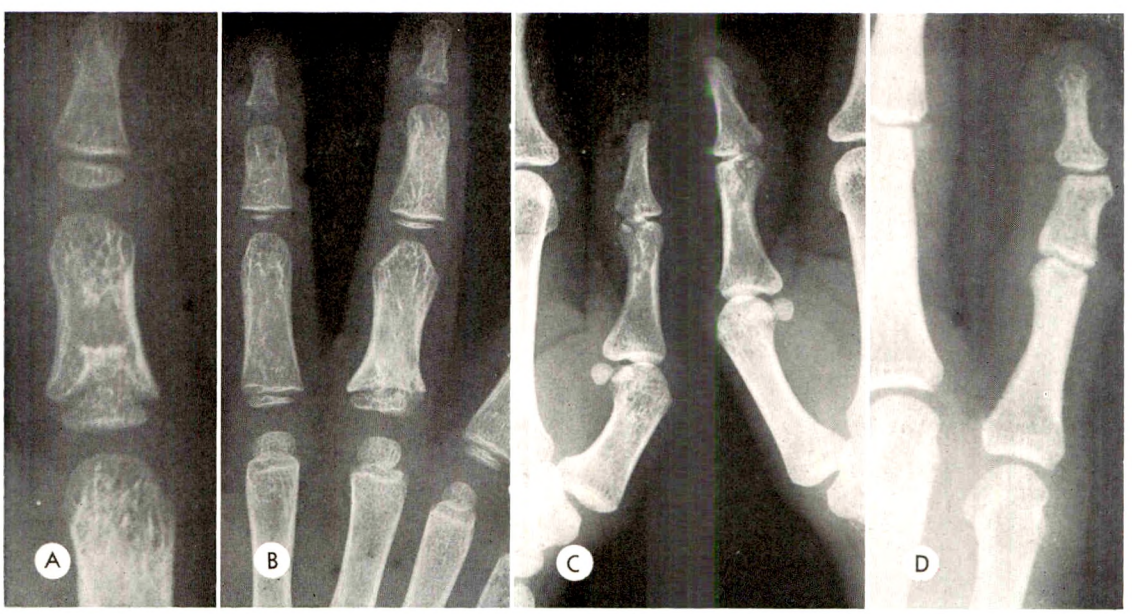


Fig. 5. Injury to the cartilage plate by diaphyseal infarction may cause growth disturbances represented by (*A*) a triangular epiphysis; (*B*) a broader shorter bone with a flared out misshapen metaphysis. When growth has ceased, the bones remain small and broad (*C* and *D*). As a rule the joint space is not disturbed.

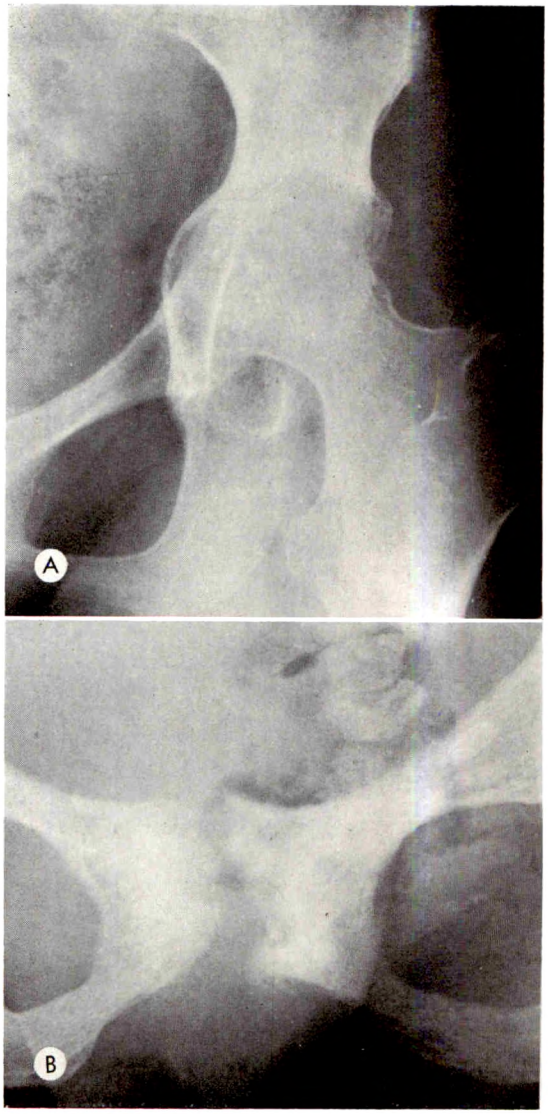
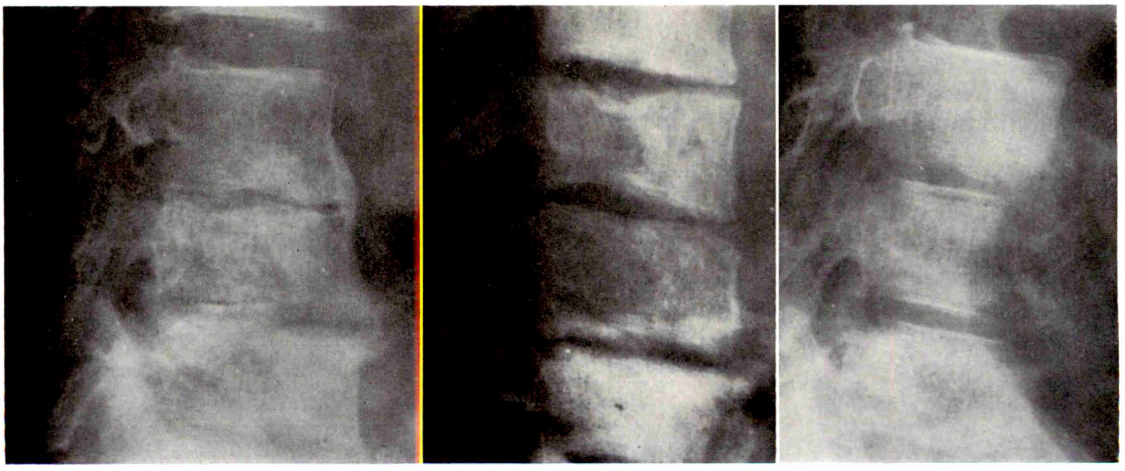


FIG. 6. (*A*) Hip joint following *Salmonella* arthritis. (*B*) Pubic osteitis and symphysitis due to *B. coli*.

deformed, characteristically in a triangular or rhomboidal fashion. It appears that the peripheral parts of the epiphyseal cartilage recover more rapidly than the central portions, probably owing to variations in the local blood supply between the two zones. A short finger is the relic of this epiphyseal damage (Fig. 5, *A–D*). The hands are good indicators of this type of change and its aftermath. Frequently, the condition is symmetric and implicates several bones.

Among the antenatal patients, 3 were found who had had dactylitis and one patient showed active bone infection. The hand roentgenogram of 2 of the 7 blood donors showed evidence of a previous dactylitis. In the clinical group, as is to be expected since there were more children, the incidence of active bone infection or infarction was far higher. Twenty-eight per cent showed evidence of one or another of these conditions and in 24 per cent the lesions were in an active stage when first seen. Five patients had active dactylitis,

FIG. 7. Lumbar spondylitis of obscure nature found in 3 women. Probably due to low grade infection.



all children under the age of ten. Three others were incidentally noted to have a shortened digit consequent on previous infarction. Of other long bones involved, the humerus was implicated 7 times, the femora 4 times, and the ulnae, tibiae and radii 3 times. Other sites affected include the clavicle, pubis and ilium. *Salmonella* bone infection frequently follows upon bone infarction in the sickle hemoglobinopathies.[8] Hendrickse and Collard[8] in their series found 4 proved instances, including an acute arthritis affecting a hip joint. The roentgenographic appearances in our cases were not sufficiently characteristic for this form of infection to be distinguished from other types of osteitis (Fig. 6, *A* and *B*).

In 3 patients with backache, changes were noted in the lumbar vertebral bodies. The patients affected were females aged sixteen, twenty-one and twenty-seven. The process in each case involved several lumbar bodies and appeared to begin in the intervertebral disk. The intervertebral disk space became diminished and this was accompanied by erosion of the adjacent vertebral bodies. Gradually repair took place with sclerosis. In 2 of the patients a low grade fever was present but there never was any significant leukocytosis. *Salmonella* and *Brucella* antibodies were not raised. The intervertebral disk is a relatively avascular structure in the normal person so it is difficult to believe, on anatomic grounds, that the disease could begin in a disk as a result of infarction. The involvement of a segment of the vertebral column is also difficult to explain on a pathologic basis. Though we are uncertain of the nature of the change here described, we believe that it probably represents infarction with later development of low grade infection (Fig. 7).

Back pain is common in sickle cell anemia, and in hemoglobin SC disease it is well recognized.[4,17,19] Backache is said to be frequent when pregnancy is present, according to Curtis.[4] It has also been suggested that a possible explanation for the sudden death in pregnancy that occurs in SC disease may be related to vertebral and other bony infarction accompanied by marrow and fat embolism.[12]

SUMMARY

The genetic basis and the clinical and hematologic features of hemoglobin SC disease are described. The roentgenographic findings in 117 Nigerian patients are reported. These consist of changes consequent on marrow hyperplasia, bone infarction and bone infection. The evolution of characteristic features in certain sites is described.

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VOL. 88, NO. 3

PINEAL TERATOMA—BEFORE AND AFTER DENTITION*

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ROENTGENOGRAPHIC interpretation is dependent upon contrasting shadows of structures. When these are characteristic of familiar objects, the diagnosis can be made with ease and certainty as in the case of an intracranial neoplasm containing a tooth. However, when the tooth is small, its shadow may be mistaken for a nonspecific intracranial calcification as illustrated by the following case.

REPORT OF A CASE

A male, aged three, was well until February, 1955, when he developed anorexia, vomiting, weight loss, and changes in mentation. On admission to Letterman General Hospital on March 12, 1955, he was found to have spontaneous nystagmus on lateral gaze and paralysis of upward gaze, but no other abnormal neurologic signs. Skull roentgenograms were negative. Ventriculograms (Fig. 1, *A* and *B*) showed symmetric dilatation of the lateral ventricles and of the anterior third ventricle with no filling distally. A pinealoma was suspected and a right ventriculocisternostomy was done on March 14, 1955. The patient improved and remained well until March, 1959 when he again developed anorexia, vomiting, and changes in mentation.

His second admission to Letterman General Hospital was on May 4, 1959. Skull roentgenograms (Fig. 2, *A* and *B*) showed scattered calcifications in the pineal region. The patient was treated with external irradiation, receiving 2,600 rads tumor dose through right and left lateral 10×10 cm. portals in twenty-nine days, (220 kv., half value layer 1.33 mm. Cu) and slowly improved.

The remission of his symptoms lasted one year and, in spite of another course of external irradiation in May, 1960, consisting of 2,600 rads tumor dose through three midline sagittal 8×10 cm. portals in thirty-nine days (280 kv., half value layer 2 mm. Cu), his condition

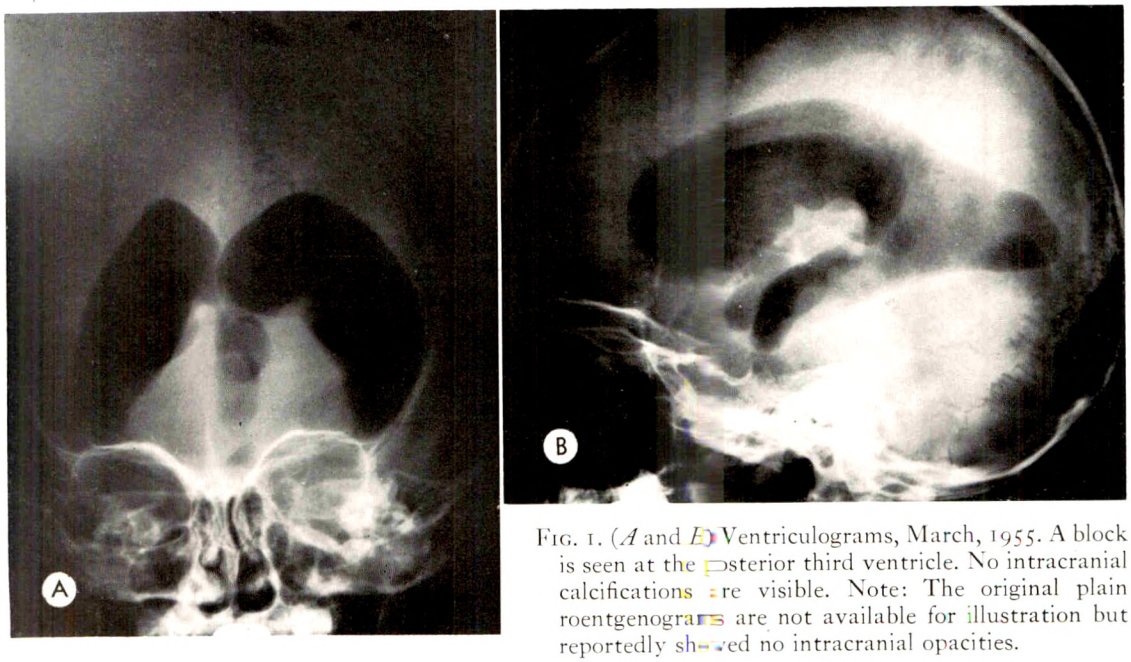


FIG. 1. (*A* and *B*) Ventriculograms, March, 1955. A block is seen at the posterior third ventricle. No intracranial calcifications are visible. Note: The original plain roentgenograms are not available for illustration but reportedly showed no intracranial opacities.

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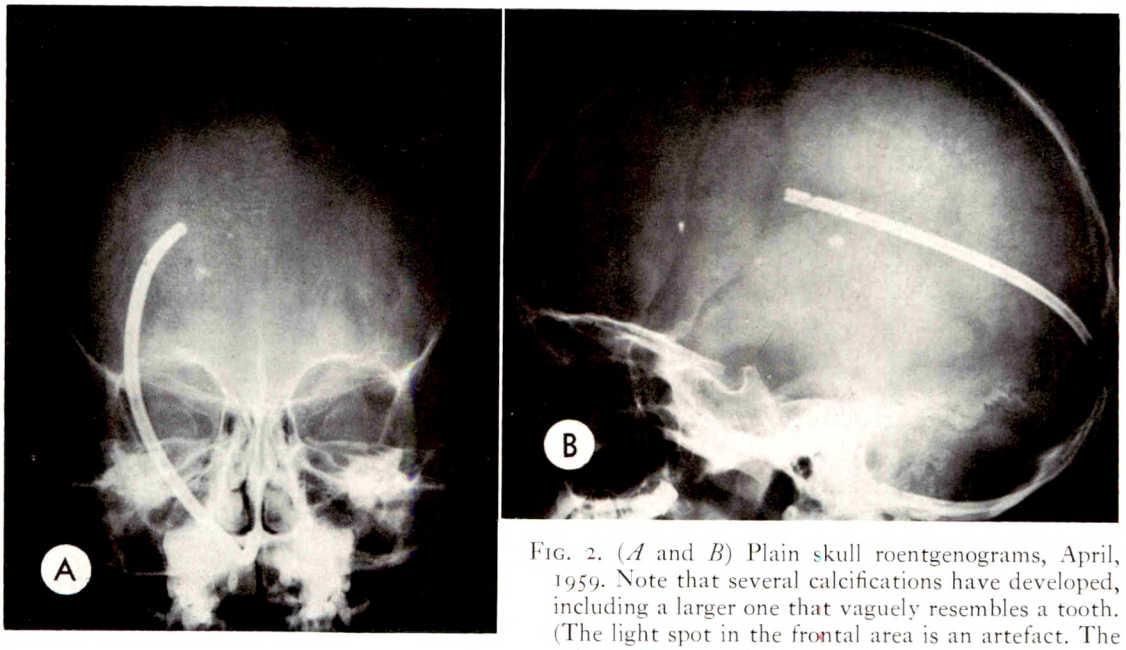


Fig. 2. (*A* and *B*) Plain skull roentgenograms, April, 1959. Note that several calcifications have developed, including a larger one that vaguely resembles a tooth. (The light spot in the frontal area is an artefact. The tube is from a Torkildsen procedure done in 1955.)

worsened. Skull roentgenograms on November 2, 1960 (Fig. 3, *A* and *B*) revealed an increase in the size and in the number of the calcifications in the pineal region, one of which was recognized to be a tooth. In retrospect, the tooth was present on the skull roentgenograms of 1959 (Fig. 2, *A* and *B*). Total excision of a tumor 6 cm. in diameter was performed on November 3, 1960. The patient survived the operation but was left with a severe neurologic impairment which has remained stationary.

The histopathologic diagnosis was teratoma, benign, containing segments of ureter, intestine, smooth muscle, brain, hair, amorphous calcium deposits (Fig. 4) and a "baby molar tooth" (Fig. 5).

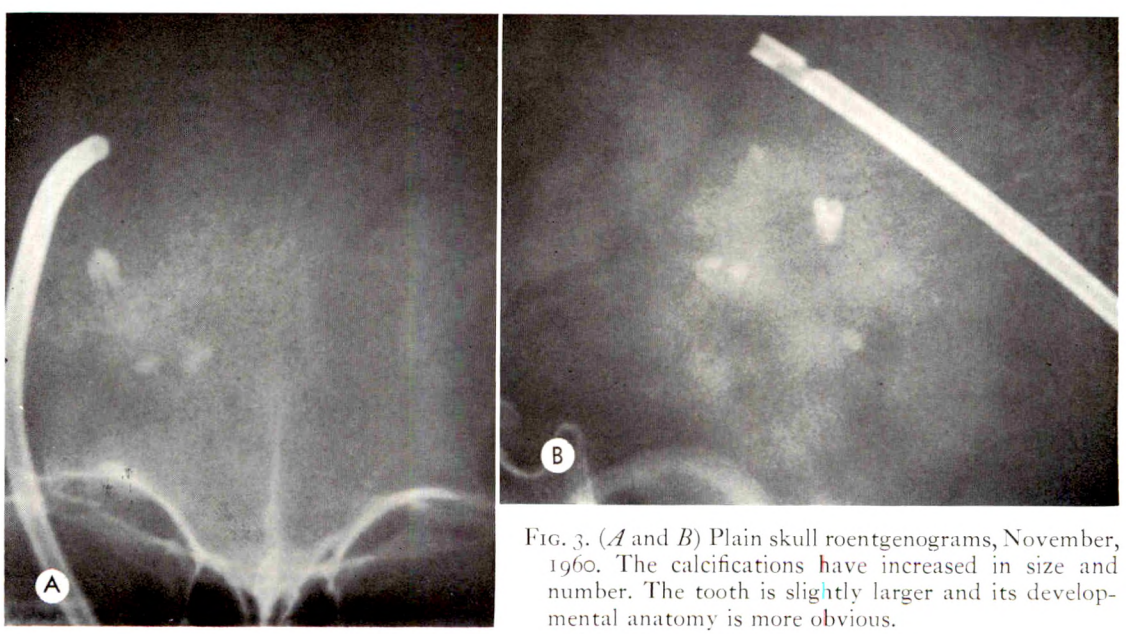
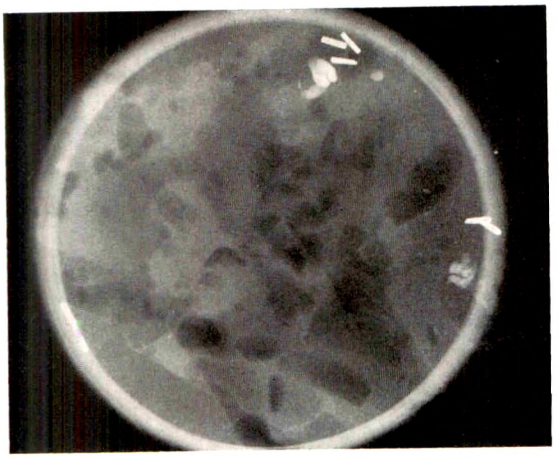


Fig. 3. (*A* and *B*) Plain skull roentgenograms, November, 1960. The calcifications have increased in size and number. The tooth is slightly larger and its developmental anatomy is more obvious.



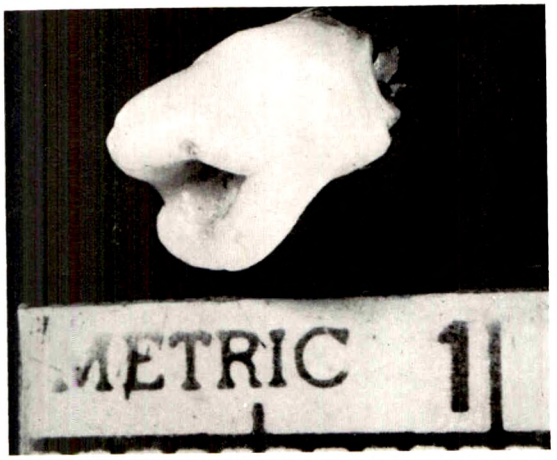


Fig. 4. Roentgenogram of the surgical specimen in a wax box. The tooth is clearly seen (upper right). Note its characteristic layers. The other calcifications are amorphous.

CONCLUSION

Teratomas of the pineal region sometimes contain a diagnostic feature which permits their preoperative differentiation from other tumors of this area.[1,2] Such a case containing a tooth is reported.

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Fig. 5. Photograph of the "baby molar" removed from the teratoma. (The scale is in centimeters.)

CYSTIC ENLARGEMENT AND OBSTRUCTION OF THE FOURTH VENTRICLE FOLLOWING POSTERIOR FOSSA SURGERY*

THE POSTOPERATIVE DANDY-WALKER SYNDROME

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TUMORS occurring in the narrow confines of the posterior fossa are well known causes of obstructive hydrocephalus. Unfortunately, surgical procedures in this area may produce arachnoiditis which may in time lead to a new blockade of the cerebral spinal fluid pathways. Although postoperative arachnoiditis is a thoroughly recognized entity, subsequent cystic transformation of the fourth ventricle due to surgical porencephaly and associated arachnoiditis has not been well documented.

In the last several years 3 patients who received surgical treatment at this institution for posterior fossa tumors have presented cystic alteration of the fourth ventricle on follow-up ventriculograms. All 3 patients exhibited recurrent symptoms of internal hydrocephalus and all 3 prior to roentgenologic examination were suspected of having recurrent tumor as the primary cause of obstruction. The similarity in the anatomic changes presented by these patients and those with enlargement of the fourth ventricle produced by congenital atresia of the foramina of Magendie and Luschka (Dandy-Walker syndrome) has been noted. A case of the latter type is included in this report for comparison.

REPORT OF CASES

CASE I (Fig. 1, *A–C*). J.B., a 3 year old male, presented with headache, unsteady gait, lethargy and vomiting. A large midline cystic cerebellar astrocytoma was partially removed at an initial operation and the entire tumor was excised at a second operation 2 weeks later. The patient returned 3 months later with signs of increased intracranial pressure. A ventriculogram (Fig. 1*B*) showed a small quantity of air in the cisterna magna indicating that a communicating hydrocephalus was prsent. A large cystic structure of the posterior fossa was interpreted as a combination of dilated fourth ventricle and a postoperative surgical porencephalic cyst containing fluid under tension. A right ventriculojugular shunt was performed and the child has remained asymptomatic during a 12 month follow-up period.

CASE II (Fig. 2, *A* and *B*). L.F., a 14 year old female, presented with signs and symptoms of a posterior fossa tumor. Ventriculograms showed obstruction in the fourth ventricle. A posterior fossa craniotomy was done with removal of a cerebellar astrocytoma which was situated predominantly on the left, but extended also to the right. The patient did not do well postoperatively and had to be reexplored for decompression of herniation of the cerebellar tonsils. Six months later the patient returned to the hospital with signs of increasing intracranial pressure. Clinical examination revealed a bulging soft tissue mass in the region of the previous craniotomy. A ventriculogram (Fig. 2*A*) showed internal hydrocephalus and a large posterior fossa cyst communicating with the aqueduct of Sylvius. No subarachnoid air could be identified. The posterior fossa was reexplored. The patient was found to have an adhesive arachnoiditis as well as a large porencephalic cyst which appeared to be a direct enlargement of the fourth ventricle. Four days following operation the patient expired. Marked cystic enlargement of the fourth ventricle was found. Several histologic sections of the cerebellum revealed the presence of tumor in the wall of the fourth ventricle. The tumor did not, however, appear to have been the primary cause of obstruction.

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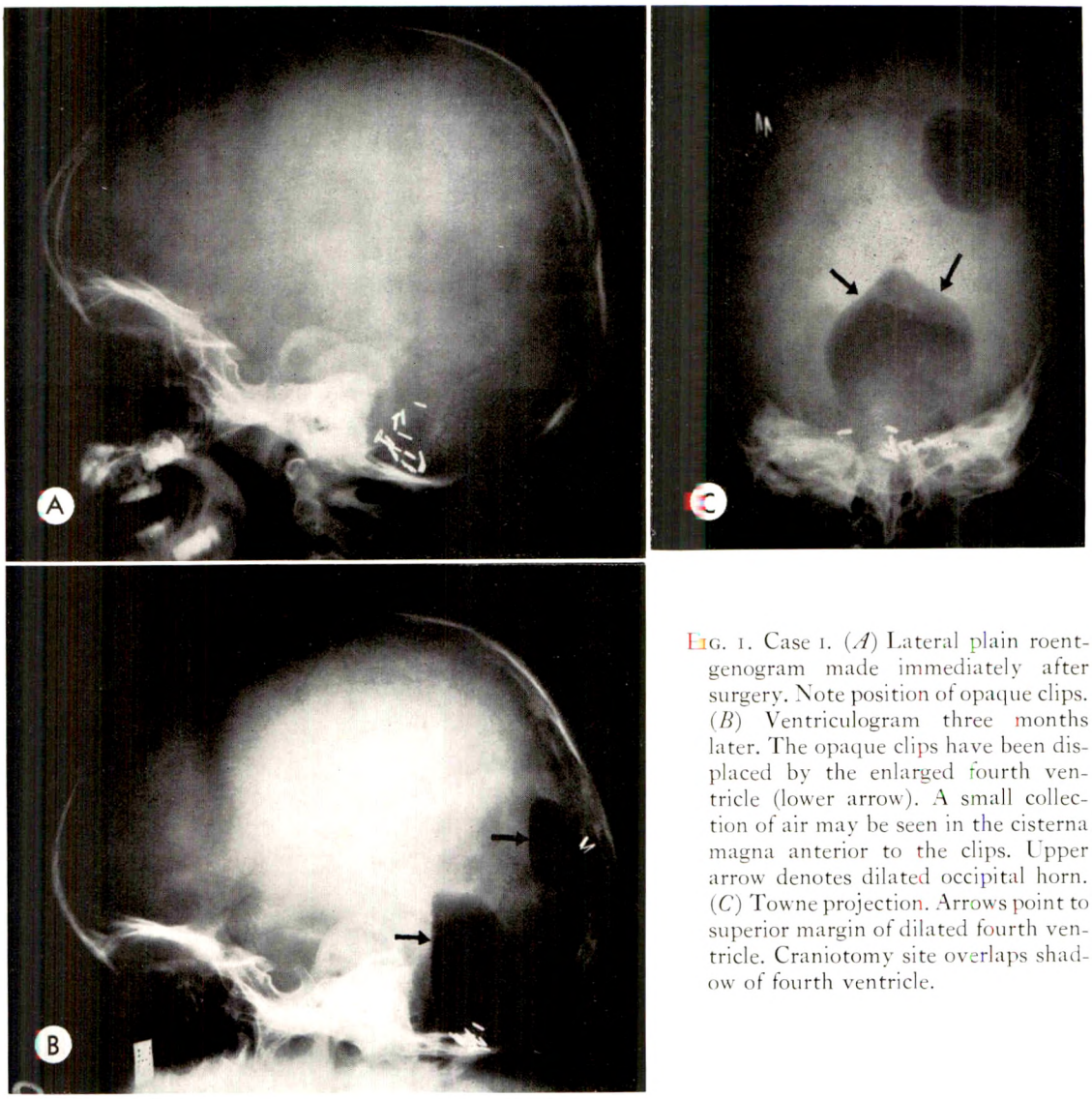


Fig. 1. Case 1. (*A*) Lateral plain roentgenogram made immediately after surgery. Note position of opaque clips. (*B*) Ventriculogram three months later. The opaque clips have been displaced by the enlarged fourth ventricle (lower arrow). A small collection of air may be seen in the cisterna magna anterior to the clips. Upper arrow denotes dilated occipital horn. (*C*) Towne projection. Arrows point to superior margin of dilated fourth ventricle. Craniotomy site overlaps shadow of fourth ventricle.

Case III (Fig. 3, *A–C*). G.T., a 52 year old female, presented with a 3 year history of signs and symptoms indicating left eighth nerve tumor. Surgical removal of the tumor was attempted; however, only partial removal was accomplished due to hemorrhage at the time of surgery. She returned 2 years later because of nausea, vomiting and difficulty in swallowing. A further intracapsular removal of the neurinoma was done. The symptoms persisted in the postoperative state and the area was re-explored with further removal of tumor as well as its capsule. At the time of the surgical procedure, hemorrhage occurred in the left cerebellar hemisphere causing an intracerebellar hematoma which required evacuation of at least 30 cc. of blood. She remained improved for 2 years but returned again because of left facial pain, nausea and vomiting. Ventriculograms showed dilatation of the entire ventricular system. A large cyst in the left posterior fossa communicating with the fourth ventricle was found. Re-exploration disclosed a large, thin-walled, arachnoid cyst in the left cerebellar hemisphere, causing compression of its remnants. Under the floor of the cyst, a recurrent tumor was found measuring approximately 4 cm. in diameter. The tumor and cyst were removed and a wide communication was made between the cisterna magna and the

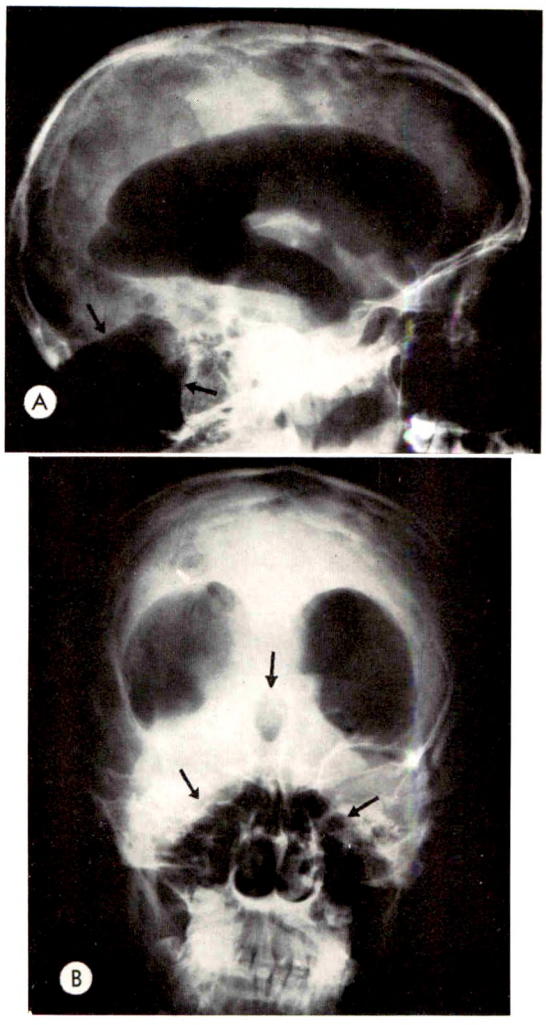


FIG. 2. Case II. (*A*) Arrows point to cystic fourth ventricle. Note protrusion of ventricle through craniotomy. (*B*) Lower arrows denote lateral margins of fourth ventricle. Upper arrow points to dilated third ventricle.

cavity on the left to prevent recurrence of cyst formation. The patient has shown continued improvement since the operation.

CASE IV (Fig. 4, *A–D*). B.D., a 3 month old female, showed evidence of hydrocephalus clinically and roentgenographically. An initial ventriculogram was mistakenly interpreted as showing communicating hydrocephalus. Right ventriculojugular, left ventriculojugular and right ventricular pleural shunts were performed at the age of 3 months, 7 months and 8 months, respectively, without success. A second ventriculogram showed marked dilatation of the lateral ventricles. The fourth ventricle could not be filled with air. Re-examination of the first ventriculogram with interpretation of the findings as indicated in Figure 4 was made. At the age of 11 months the child had a posterior fossa exploration and a large cystic fourth ventricle compatible with the Dandy-Walker syndrome was found. The patient expired during this procedure.

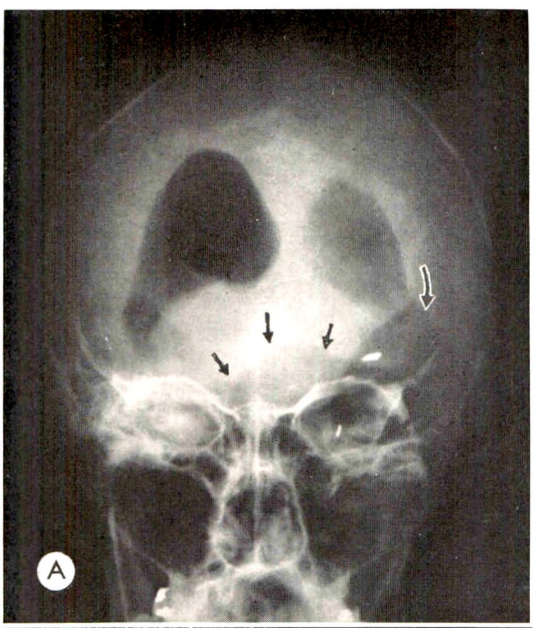
DISCUSSION

The propensity of the spinal and cerebral meninges to form inflammatory cysts which in turn produce tumor-like symptomatology has been recorded intermittently over a 50 year period. Horsley[3] in 1909 indicated that arachnoidal collections of fluid were a more common cause of spinal cord dysfunction at that time than extramedullary tumors. The adverse effects of inflammatory alteration of the posterior fossa meninges were recorded by Horrax[4] in 1924. He presented a series of 33 patients in whom acute and chronic cisternal arachnoiditis had been found without evidence of tumor at the time of surgical exploration. Apparently the majority of operations revealed not only a thickened arachnoid membrane but a dilated cisterna magna, which as the author stated "contained pent-up cerebral spinal fluid under pressure." It is of interest here that the presumptive diagnosis prior to surgery was tumor, and that at the time of his account 28 of 33 patients surviving operation had remained well or were improved for a 1 to 9 year period. The evolution of antibiotic and steroid therapy has no doubt made meningitis a less common complication when bacterial invasion occurs in other body organs. Certainly the incidence of arachnoidal cyst formation on an inflammatory basis would appear to be reduced now as compared with that prior to 1940.

Of interest is the report by Handa and Bucy[3] of a benign cyst found in the vermis of the cerebellum. The cyst did not communicate with the fourth ventricle or subarachnoid space but presented as a posterior fossa tumor producing obstructive

symptomatology. No evidence of inflammatory change was found and the lesion was thought to be a congenital anomaly. Another variation of congenital benign cystic change has been pointed out by Gardner *et al.*[2] They felt that separation of remnants of the embryonic rhombic roof allowed the development of arachnoid cysts in 4 of their patients and that this entity differed from the true cystic alteration of the fourth ventricle which is seen in patients with the Dandy-Walker syndrome.

Trauma, with tearing of the meninges and subsequent bleeding into the subarachnoid space, remains an important cause of arachnoiditis. Thompson[7] in 1946 and later Nichols and Manganiello[6] reported cases of arachnoidal cyst formation in the posterior fossa which could be ascribed to antecedent trauma. In Thompson's patient an arachnoid cyst developed in the region of the cisterna magna. Progressive symptoms due to obstructive hydrocephalus and local pressure effects caused by the cyst were noted over a two year period following injury. Air studies confirmed the presence of hydrocephalus with evidence of obstruction at the fourth ventricle level. Surgical re-





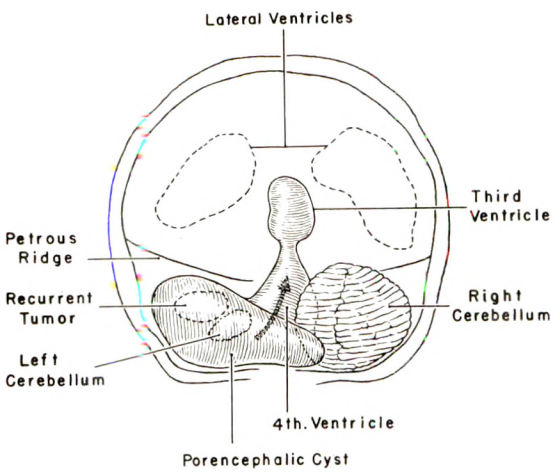


Fig. 3. Case III. (*A*) Air filled cyst in left posterior fossa indicated by lower arrows. Upper arrow points to craniotomy. (*B*) Dotted line delineates enlarged fourth ventricle. (*C*) Diagrammatic representation of roentgenographic and surgical findings. The skull is viewed as seen from an occipital approach at surgery.

moval of the cyst resulted in return of normal neurologic function.

Arachnoidal scarring secondary to surgical trauma is a necessary risk of posterior fossa surgery. Although meticulous surgical dissection and hemostasis may be carried out, trapped cerebral spinal fluid, blood and residual tumor may remain. These factors and inflammatory reaction to them serve as a basis for subsequent obstruction in the posterior fossa. The production of large tissue defects following the removal of tumor or normal brain substance creates a ready made space for an expanding collection of fluid. Communication of the tissue defect with the fourth ventricle is often established at the time of surgery. Subsequent adhesive encasement of the outlet foramina of the fourth ventricle may produce marked cystic alteration of this structure.

Porencephaly caused by trauma, vascular occlusion, or a developmental abnormality may be associated with arachnoiditis. A porencephalic cyst of this type, however, rarely traps fluid under pressure. Impeded drainage of cerebrospinal fluid from an area of surgical porencephaly when it occurs would seem to be primarily on the basis of surrounding inflammation. The

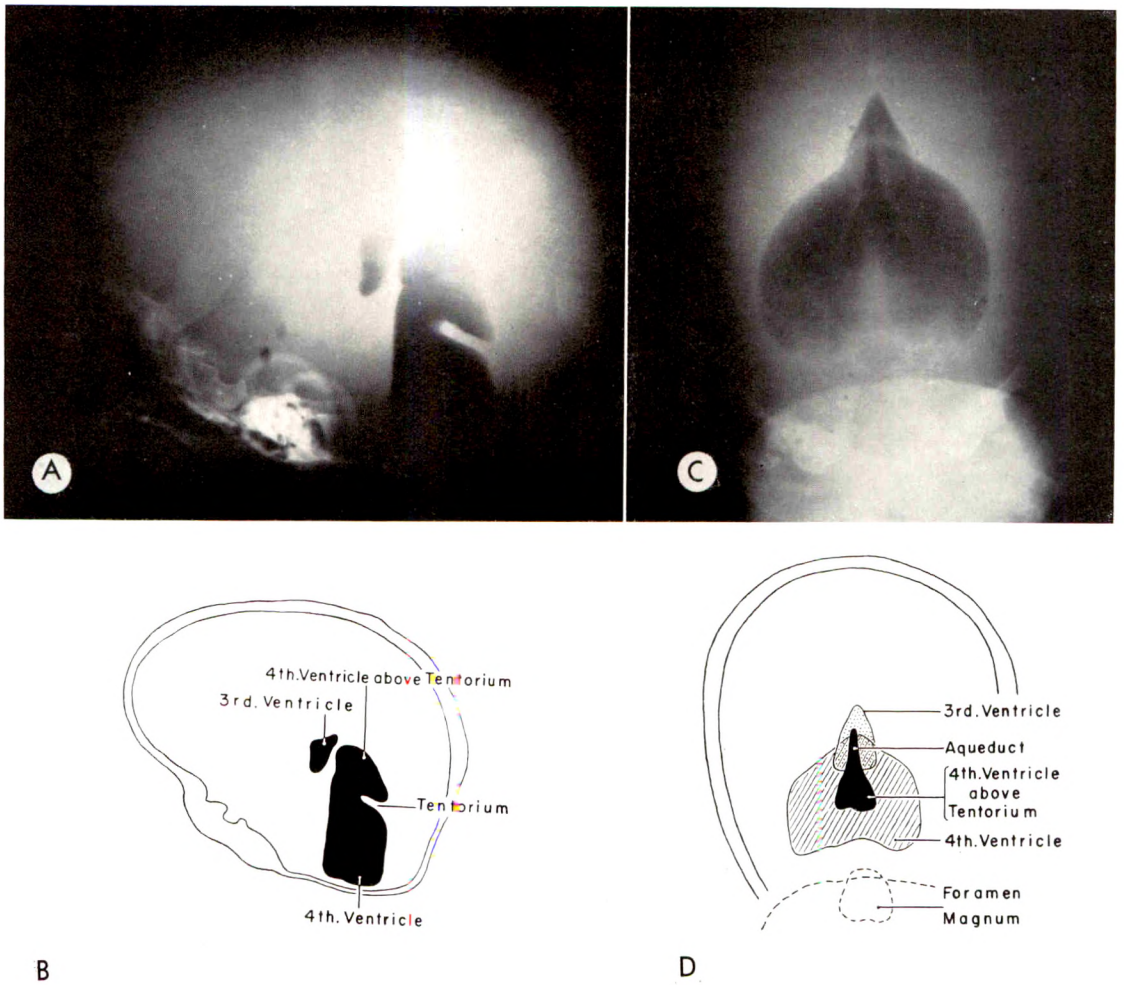


FIG. 4. Case IV. Dandy-Walker syndrome. (*A*) Lateral ventriculogram taken after filling of third and fourth ventricles. (*B*) Diagram of lateral projection. (*C*) Towne projection. (*D*) Diagram of structures seen in the reverse-Towne projection.

degree of obstruction of the porencephalic cyst produced by residual tumor is difficult to determine. Apparently a direct sealing of the outlets of the fourth ventricle by tumor in Cases II and III did not occur. An inflammatory response due to residual tumor, however, cannot be excluded.

Knowledge of pre-existing alteration of brain structure and also of the details of post-surgical procedures can be invaluable in both clinical and roentgenographic evaluation of patients with recurrent hydrocephalus after excision of posterior fossa tumors. In the first 3 patients described surgical manipulation had produced a real or potential area of porencephaly. The fact that a large cystic astrocytoma had been removed in the second patient explained in part the enlargement of the fourth ventricle. In the third patient the evacuation of fluid from an intracerebellar hematoma at surgery with the associated loss of brain substance serves as an explanation for the presence of a large air-filled structure in the left side of the posterior fossa as seen on the roentgenographic studies.

The first case illustrates how roentgenographic visualization of cystic dilatation may influence the type of therapeutic measures employed. Although the presence of tumor was not completely ruled out after the air study, the patient's symptoms could be satisfactorily explained on the basis of cystic dilatation and partial inflammatory obstruction of the fourth ventricle foramina. The establishment of a bypass shunt has, at least for the present, alleviated the condition and obviated a second surgical exposure of the posterior fossa.

The second patient highlights a problem in the postoperative differential diagnosis of soft tissue swelling adjacent to the occipital bone defect. A meningocele produced on a surgical basis is a not uncommon aftermath of posterior fossa surgery. If surgical repair is needed, the indications are usually primarily cosmetic. The air study in this patient confirmed the suspicion that the problem was of greater complexity than the presence of a meningocele alone. Although herniation of a cystic structure had occurred, it was in direct communication with the ventricular system and had produced an obstructing hydrocephalus.

A comparison of the first 3 cases with 1 presenting the Dandy-Walker syndrome has been made principally to emphasize the resemblance of the mechanical effects of outlet obstruction of the fourth ventricle seen in both. Although Case IV would appear to be only a moderate example of dilatation of the fourth ventricle, enough fluid under pressure had collected to cause herniation of the ventricle above the tentorium. Many of the cases reported in the literature including those of Dandy[1] have shown massive enlargement of the fourth ventricle. It is readily understandable how a ball of fluid occupying the entire posterior fossa can effect thinning and bulging of the occipital bones and cause upward displacement of the transverse sinuses. In the postoperative state, on the other hand, local pressure effects of this type would not be expected due to the bony exit offered by the previous craniotomy and the resistance of the older calvarium in which they may be found.

Marked dilatation of the lateral ventricles is a common sequel to fourth ventricle obstruction in the Dandy-Walker syndrome. Ventricular dilatation may persist in patients after removal of posterior fossa tumors without evidence of recurrent obstruction. The degree of ventricular enlargement in the cases reported due to residual preoperative dilatation or recurrent obstruction in the posterior fossa is difficult to assess. The early recognition of a return of increased cerebral spinal fluid pressure in the first 3 patients is the probable reason why the lateral ventricles in postoperative cystic obstruction did not achieve a gigantic size.

SUMMARY

Three cases of cystic alteration of the fourth ventricle following posterior fossa surgery with associated hydrocephalus have been described. Removal of normal

and neoplastic cerebellar tissue as well as intracerebellar hematomas may serve as initiating factors in cyst formation. An associated arachnoiditis due to postoperative inflammation or residual tumor may complete the formation of the cyst which then becomes an obstructive reservoir for cerebral spinal fluid. A similarity in disturbance of cerebral spinal fluid mechanics produced by postoperative cysts and the cystic change of the fourth ventricle in the Dandy-Walker syndrome has been noted.

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BRONCHIAL ADENOMA IN CHILDREN

A REVIEW OF THE LITERATURE AND REPORT OF THREE CASES

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PRIMARY bronchial neoplasms are uncommon in the pediatric age group. Bronchogenic carcinoma has been reported rarely in children.[5,17,28,29,39,60,72] Bronchial adenoma occurs with greater frequency than is usually considered and is one of the more common tumors in this group. This is in contrast to the incidence of the two entities in adults where bronchial adenoma represents approximately 6 per cent of all primary bronchial neoplasms.

Suter[63] reviewed the literature in 1952 and reported 12 cases of primary bronchogenic carcinoma in children under twelve years of age. It is possible that some of these cases represent what today would be histologically recognized as bronchial adenoma. In addition Beardsley,[5] Kanof,[29] and Wasch *et al.*[72] have each reported a case of carcinoma of the lung in a child under twelve.

In our search of the literature we have found 16 cases of proved bronchial adenoma in children twelve years of age or under, and in addition we are reporting 3 cases for a total of 19. Sixteen other cases have been reported between the ages of twelve and sixteen (Table 1).

With recent advances in thoracic surgery, the prompt recognition of these tumors is becoming increasingly important in order to prevent serious pulmonary complications such as recurrent pneumonitis, atelectasis, lung abscesses, bronchiectasis and empyema. In addition to the above complications, the potential malignant capabilities have been well demonstrated[1,19,41,77] and this makes early diagnosis and the proper treatment of the utmost importance.

GENERAL CONSIDERATIONS

Although Müller[44] is credited with the original description of a bronchial adenoma at necropsy in 1882; Laennec[33] in 1831 reported a vascular bronchial polyp which probably represented the same entity. Between these earliest reports and 1930 there was much confusion in naming and reporting the various benign and low-grade malignant neoplasms of the bronchial tree. Goldman and Conner[19] presented an extensive historical review of the literature prior to 1950 and stated that Horn described a bronchial adenoma in 1907. Lowry and Rigler[34] credited Von Eicken with the bronchoscopic removal of what probably was an adenoma in the same year. Jackson[24] in 1917 reported bronchoscopic removal of an endothelioma of the right bronchus in a thirty-five year old male; this also probably represented a bronchial adenoma. Reisner[49] in 1928 described a typical adenoma in a forty-seven year old woman and briefly reviewed the German literature.

In 1930 Dr. Rudolph Kramer[31] presented a paper in Atlantic City at the Thirteenth Annual Meeting of the American Bronchoscopic Society discussing the clinical findings in 2 cases of bronchial adenoma which were treated by bronchoscopic removal and implantation with radon seeds. In 1932 Wessler and Rabin[75] summarized their clinical experience with 12 cases in their classic paper; these authors believed at that time that bronchial adenomas might undergo malignant change.

Peterson[48] in reviewing the literature in 1936, could find only 43 reported cases. Since that time several hundred bronchial

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TABLE I

SUMMARY OF CASE REPORTS OF BRONCHIAL ADENOMA IN CHILDREN

	Age (yr.)	Sex	Side	Type	Year Reported	Reported by
1.	4	M	L	*	1953	Berger *et al.*[6]
2.	6	F	L	Carcinoid	1954	Roberts[50]
3.	7	M	L	*	1954	Ward *et al.*[70]
4.	8	F	R	*	1945	Barrett and Barnard[4]
5.	8	F	L	*	1958	Wawrykowicz[73]
6.	8	F	L	*	1947	McReynolds and Parrish[36]
7.	9	M	L	*	1958 1959	Vawter and Ferguson[68,69] Farber and Craig[14]
8.	9	F	R	*	1951	Smoller and Maynard[57]
9.	9	F	R	Cylindroma	1956	Sherman *et al.*[55]
10.	9	F	L	Carcinoid	1956	Sherman *et al.*[55]
11.	10	F	L	Carcinoid	1950	Goldman and Conner[19]
12.	10	F	R	*	1954	Hallman and Turunen[21]
13.	10	F	*	Carcinoid	1954	Soutter *et al.*[61]
14.	11	M	R	Carcinoid	1961	Present report
15.	11	F	R	Carcinoid	1961	Present report
16.	11	F	L	*	1961	Present report
17.	11	M	*	*	1936	Peterson[48]
18.	11	F	R	*	1954	Thomas[64]
19.	12	F	L	*	1955	Ferraris and Metras[16]
20.	13	F	Bifurcation	*	1956	Crafoord and Lindgren[9]
21.	13	M	L	Carcinoid	1950	Goldman and Conner[19]
22.	13	M	R	Carcinoid	1945	Jackson *et al.*[26]
23.	13	F	*	*	1935	Kramer and Som[32]
24.	13	M	L	Carcinoid	1949	Souders and Kingsley[59]
25.	14	F	L	Clear cell	1950	Goldman and Conner[19]
26.	14	M	L	*	1958	Schroder[54]
27.	14	M	R	*	1944	Lowry and Rigler[34]
28.	15	F	R	Carcinoid	1945	Jackson *et al.*[26]
29.	15	F	R	Carcinoid	1945	Jackson *et al.*[26]
30.	15	M	*	*	1950	Moersch and McDonald[43]
31.	15	M	R	Carcinoid	1956	Sherman *et al.*[55]
32.	16	F	L	Solid	1950	Smidt[56]
33.	16	F	R	*	1947	Maier and Fischer[37]
34.	16	F	R	Carcinoid	1950	Goldman and Conner[19]
35.	16	M	R	*	1959	Saada and Mahassen[52]

* Not specified.

adenomas have been reported and much work has been accomplished in better defining the pathology and the natural history of the disease.

INCIDENCE

Since 1947 a total of 12 cases of bronchial adenoma has been recorded with the tumor registry at Fitzsimons General Hospital, comprising 5.27 per cent of 228 primary bronchial neoplasms seen at this institution. The incidence of the tumor cited by various other authors[8,46,47,76] has ranged from 1.2–12.0 per cent.

Bronchial adenoma occurs somewhat more frequently in the female than in the male. In a series of 387 cases of bronchial adenoma collected from the literature, 58 per cent were in females, which is in direct contrast to bronchogenic carcinoma. Fifty-four per cent of the adenomas were on the right side.

Approximately 90 per cent of the bronchial adenomas occur between the ages of

twenty and fifty. The peak of incidence is in the fourth decade, again in contrast to bronchogenic carcinoma which has a peak incidence in the sixth decade.

SYMPTOMATOLOGY

The majority of the symptoms caused by bronchial adenomas can be ascribed either to the marked vascularity or to the intrabronchial location causing partial or complete obstruction of a portion of the bronchial tree. The slow growth of the neoplasm accounts for the duration of the symptoms.

Cough is present in from 65–85 per cent of the cases. It may be either productive or nonproductive and is usually persistent over an extended period. Hemoptysis is present in from 45–83 per cent of the reported cases. It may vary from blood streaked sputum to fatal hemorrhage. Frequently, a hemorrhage starts and stops abruptly, and in the female often coincides with the menstrual period. It may occur with bronchoscopic biopsies, either during or following the procedure.

A feeling of discomfort or even frank pain is noted rather often. Fifty per cent of 26 patients reported by Delarue[11] complained of this symptom, while Moersch and McDonald[43] found it present in 38 of their 86 cases (44.2 per cent). Fever has been reported; however, it can be attributed to inflammatory processes secondary to the obstruction of the bronchial tree by the adenomas. Occasionally, a patient will be entirely asymptomatic with the lesion being found on routine chest roentgenography.

PATHOLOGIC CHARACTERISTICS

Since some of the earliest descriptions of bronchial adenomas by Kramer[31] and later by Wessler and Rabin,[75] there have been varied opinions as to the histologic origin. Hamperl[22] in 1937 was the first to describe two histologic patterns seen in bronchial adenomas. The majority of his cases exhibited a histologic pattern which had a basic resemblance to intestinal carcinoid and which he called "carcinoid variant." Another group was similar to salivary gland tumors and these he classified as "cylindromas." From this work and also from the report by Stout[62] comes the generally accepted view that these tumors arise from the duct epithelium of the bronchial mucous glands. Womach and Graham[77] felt that they develop from arrested bronchial buds which have failed to grow normally. Mallory[38] disagreed with this theory, believing that these tumors were closely related to carcinoids. The relationship to intestinal carcinoids was more recently stressed by Warner and Southern[71] and Schneckloth *et al.*[53] who had patients with bronchial adenomas (carcinoid type) with a definite carcinoid syndrome and strongly positive 5-hydroxyindole acetic acid levels. Holley[23] has been the only one to report a bronchial adenoma that has demonstrated silver-positive granules with a Fontana stain. It is currently believed that approximately 90 per cent of bronchial adenomas are of the carcinoid type, with very few of these producing the carcinoid syndrome, and approximately 10 per cent of the cylindromatous type.[42]

Early in the study of this disease, malignant potentialities were not recognized by some authors.[26,31] As an increasing number of cases was reported, it became apparent that it must be considered to be a low grade malignancy capable of direct invasion as well as metastasis. Zamora and Schuster[78] published the first case of metastases from a bronchial adenoma in 1937. This was followed by numerous other reports of distant metastases.[2,3,77] Additional support for the theory of low grade malignancy was gained from the reports of Stout[62] and Ackerman.[1] Meissner[41] found tumor positive regional lymph nodes in 20 per cent of his cases and Ochsner and David reported 20 cases of hepatic metastases. The evidence is now sufficient so that bronchial adenoma must be considered a malignant disease.

In discussing the macroscopic and microscopic characteristics, it is again necessary to subdivide these tumors into the carcinoid and cylindromatous types.

The carcinoid adenomas have a fairly typical macroscopic appearance. Usually, there is a tumor mass in the main or primary bronchi that has a conspicuous endobronchial and extrabronchial portion, either portion of which may represent the bulk of the tumor. The endobronchial portion is characteristically smooth, round, red or pink, soft, and frequently with prominent vessels on the surface. The extrabronchial portion is well demarcated and tan-pink to pale gray.

Microscopically, the epithelial covering shows squamous metaplasia. Beneath this epithelial covering is a layer of loose connective tissue with abundant vessels. This is usually the site of the profuse bleeding common in these patients. The cellular structure of the tumor mass is quite constant. The individual cells are small, cuboid to polygonal in shape, and with a round dark central nucleus. The cell size is variable with grouping into alveolar, columnar, or nodular patterns. Conspicuous chromatin stippling is common and cellular borders are often indistinct. Characteristically, there is a rarity of mitotic figures but definite microscopic evidence of invasion of the lung by small islands of neoplasm and direct extension into lymph nodes may be demonstrated.

As mentioned earlier, the cylindromatous type of bronchial adenoma constitutes approximately 10 per cent of all bronchial adenomas and presents a considerably different pathologic appearance. Macroscopically, these tumors are found, in general, in a more peripheral position in the bronchial tree. The tumor mass extends more diffusely into the bronchial wall and is pale yellow and firm. The histologic pattern bears a great resemblance to that of mixed tumors of the salivary gland and occasionally contains cartilage and bone.

ROENTGEN FINDINGS

The roentgen findings vary greatly. A few individuals will present with an essentially normal appearing chest roentgenogram. Most of the patients, however, will have significant findings which, when coupled with the symptoms, should aid in suggesting the correct diagnosis.

With an obstructing lesion of the bronchial tree, many patients will show a pneumonitis when first seen by the roentgenologist. This may be either segmental or lobar depending upon the location of the adenoma and it is frequently associated with some degree of atelectasis (Cases 1 and 11, Fig. 1, *A* and *B*; and 6, *A* and *B*). There is often recurrent pneumonitis in the same area and a careful medical record may reveal delayed clearing of the infection. Various authors[18,23,35,61] have reported pneumonitis in from 33–60 per cent of the patients with these tumors.

With repeated occurrences of slowly resolving pneumonitis, bronchiectatic changes take place. Prominent peribronchial markings seen on the plain roentgenograms of these patients with recurrent pneumonitis should cause one to suspect bronchiectasis. Bronchograms may delineate the extent of destruction of the lung and bronchi distal to the adenoma, aiding the surgeon. Knesevitch *et al.*[30] found pneumonitis and/or bronchiectasis in 15 of their 21 patients, while Soutter *et al.*[61] reported it in 33 of their 60 cases. If the intrabronchial portion of the adenoma is large enough to act as a ball-valve, then obstructive emphysema may be noted on the plain roentgenograms (Case 1, Fig. 1, *A* and *B*). As the lesion enlarges to completely fill the bronchus or when a pneumonitis sufficiently compromises the bronchial lumen, atelectasis ensues. Atelectasis was present in 18 of Delarue's[11] 26 patients; Holley[23] reported it in 41 per cent of his series and Kramer and Som[32] in 55 per cent.

Several of the cases seen at Fitzsimons General Hospital have presented with solitary nodules in the lung fields. Knesevitch *et al.*[30] reported that 7 of their 21 cases first appeared with pulmonary nodules. These are usually well defined with no surrounding reaction and they do not contain calcification, although Bluth[7] and Mallory[38] both mention adenomas containing calcification.

Laminagrams may aid in delineating the extent of the adenoma and defining its relationship to the bronchial wall. Bronchograms may reveal a rounded deformity protruding from the wall, as well as the secondary bronchiectasis.

DIAGNOSTIC PROCEDURES

In any young patient with a history of chronic cough, recurrent pneumonitis, or hemoptysis, bronchial adenoma should be considered. Roentgenologic examination may demonstrate changes that should make one suspicious of this diagnosis, particularly when supported by laminagraphic and bronchographic studies. Final diagnosis rests on bronchoscopy.

Due to its common location in the large main stem bronchi, the great majority of these tumors will be visible. Moersch and McDonald[43] saw the tumor in 78 of 84 bronchoscopies and tissue was obtained which microscopically confirmed the diagnosis in 75 of the 78 cases. The incidence of visualization varies considerably from 62 per cent by Soutter *et al.*[61] to 93 per cent by Ochsner and Ochsner[46] with most authors visualizing between 80 and 90 per cent of the tumors.[11,30]

Bronchoscopically, the typical tumor is pedunculated, bleeds easily, pink to reddish and covered by a mucous membrane. It is usually soft, smooth and slightly polypoid. Ulceration is rarely seen, although frequently there is evidence of infection. Biopsy is usually possible; however, an adequate specimen must be obtained because the superficial biopsies may contain no tumor cells but instead may show squamous metaplasia in the epithelium and nondiagnostic superficial stroma. Because of the highly vascular nature of these tumors, biopsy may lead to profuse hemorrhage and, occasionally, to death.[10,30]

REPORT OF CASES

The following 3 case reports added to those from the literature make a total of 19 cases of bronchial adenoma in children under the age of twelve (Table 1). Two of our cases had definitive surgery and today are alive and well. The third child expired seven years after the onset of symptoms and three years after definitive surgery, developing a bronchopleural fistula and metastatic carcinoma.

Case 1. This eleven year old white male entered Fitzsimons General Hospital in June, 1960 with the chief complaint of a chronic nonproductive cough of two years' duration. At the age of nine, while the patient was outside the continental United States, a diagnosis of right middle lobe pneumonitis had been made and this responded slowly to antibiotic therapy. During the ensuing two years, three more bouts of pneumonitis in the right middle and right lower lobes occurred. There was no history of foreign body aspiration nor of hemoptysis.

Physical examination on admission was within normal limits. Skin tests for tuberculosis, histoplasmosis and coccidioidomycosis were negative as were three perspiration electrolyte tests for fibrocystic disease. All sputum cultures revealed normal flora.

Roentgenograms of the chest (including laminagrams) revealed atelectasis with cystic changes of the right middle lobe (Fig. 1, *A* and *B;* and 2). There was emphysema of the right lower lobe. Bronchograms demonstrated a rounded filling defect at the level of the right middle lobe take-off; the right middle and lower lobes did not fill (Fig. 3). Since the right lower lobe was obviously aerating, the phenomenon of nonfilling of this lobe was probably secondary to the viscosity of the contrast medium (dionosil, oily).

Bronchoscopy revealed a tumor mass of the intermediate bronchus 2 cm. below the right upper lobe take-off. A biopsy was taken and the histologic diagnosis of bronchial adenoma of the carcinoid type was established. No elevation of 5-hydroxy-indoleacetic acid was found in either the serum or urine.

Preoperative roentgenogram showed incomplete clearing of the right middle lobe density (Fig. 4).

At surgery it was found necessary to sacrifice the right middle and lower lobes (Fig. 5). The diagnosis of carcinoid bronchial adenoma was verified by permanent pathologic sections. The regional lymph nodes showed no histologic evidence of the tumor. Atelectasis and bronchiectasis were present in the distal lung. The patient's postoperative course was uneventful.

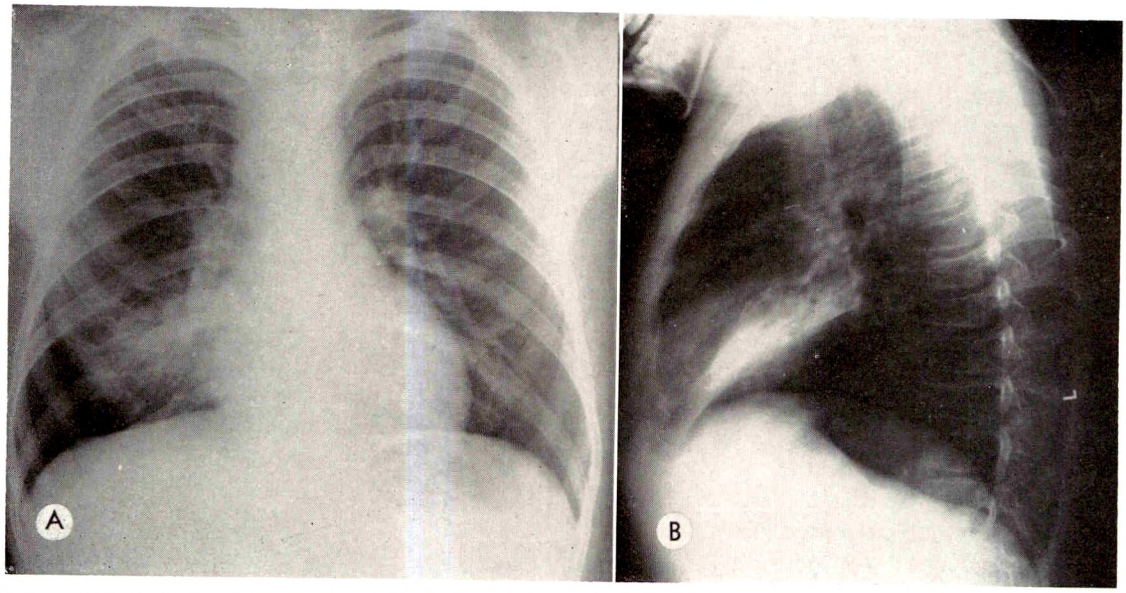


FIG. 1. Case 1. (*A*) Posteroanterior and (*B*) lateral roentgenogram of the chest early in the course of the disease showing abnormal patchy density and loss of volume of the middle lobe secondary to pneumonia and atelectasis.

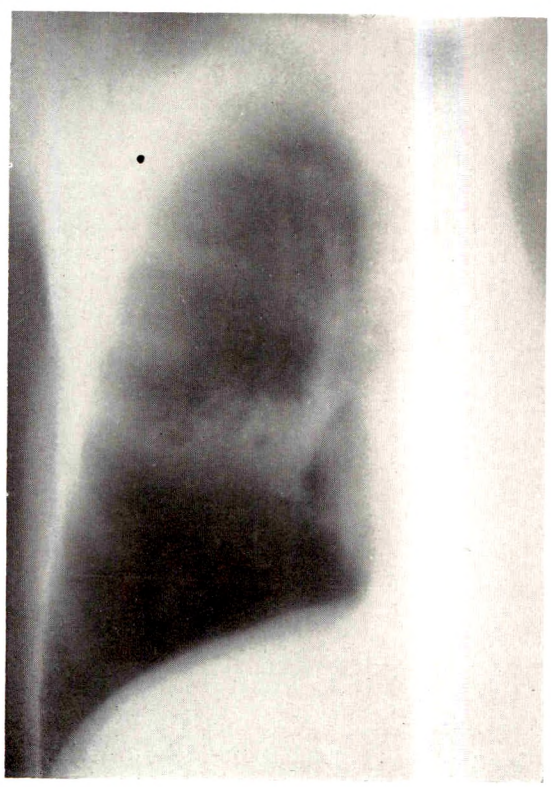


FIG. 2. Case 1. Laminagram demonstrating atelectasis of the right middle lobe and obstructive emphysema of the right lower lobe.

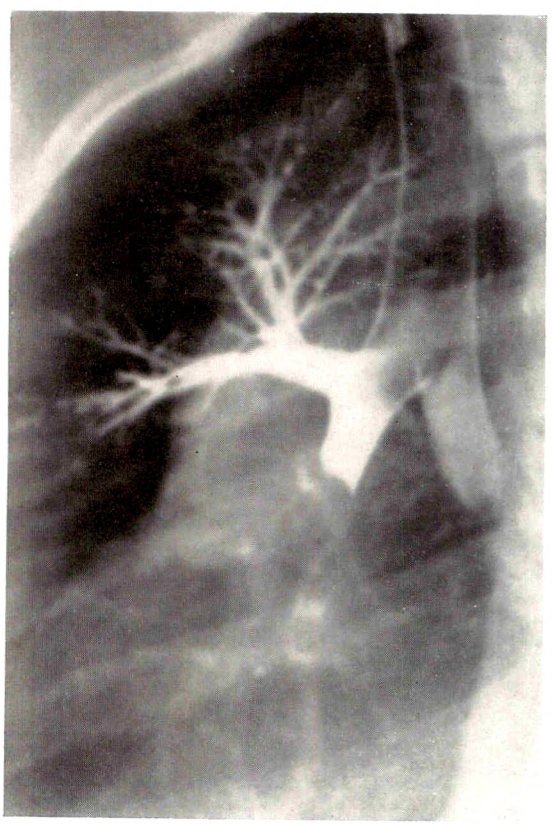


FIG. 3. Case 1. Bronchogram showing an intraluminal filling defect of the intermediate bronchus and air contrast of the right lower lobe bronchus.

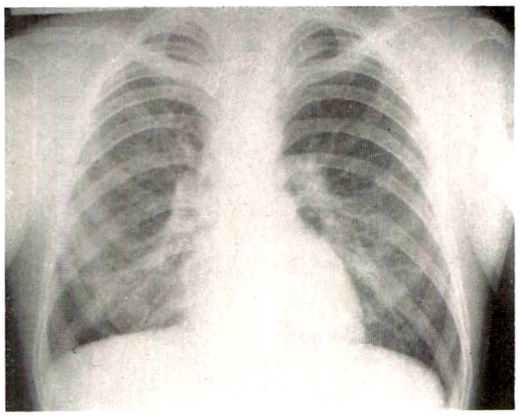


Fig. 4. Case I. Preoperative roentgenogram revealing incomplete clearing of the right middle lobe density.

Case II. M.P., a ten and one-half year old white female, was admitted to the Children's Hospital with complaints of recurrent pneumonia of the right middle lobe plus a chronic cough and intermittent colds. Past history revealed that she was first hospitalized elsewhere one year earlier for her third episode of pneumonia. Roentgenography showed abnormal density and loss of volume in the right middle lobe (Fig. 6, *A* and *B*). This cleared slowly with treatment. There were two more admissions in the ensuing year with episodes of fever, cough



Fig. 5. Case I. Surgical specimen illustrating polypoid endobronchial tumor of the intermediate bronchus.

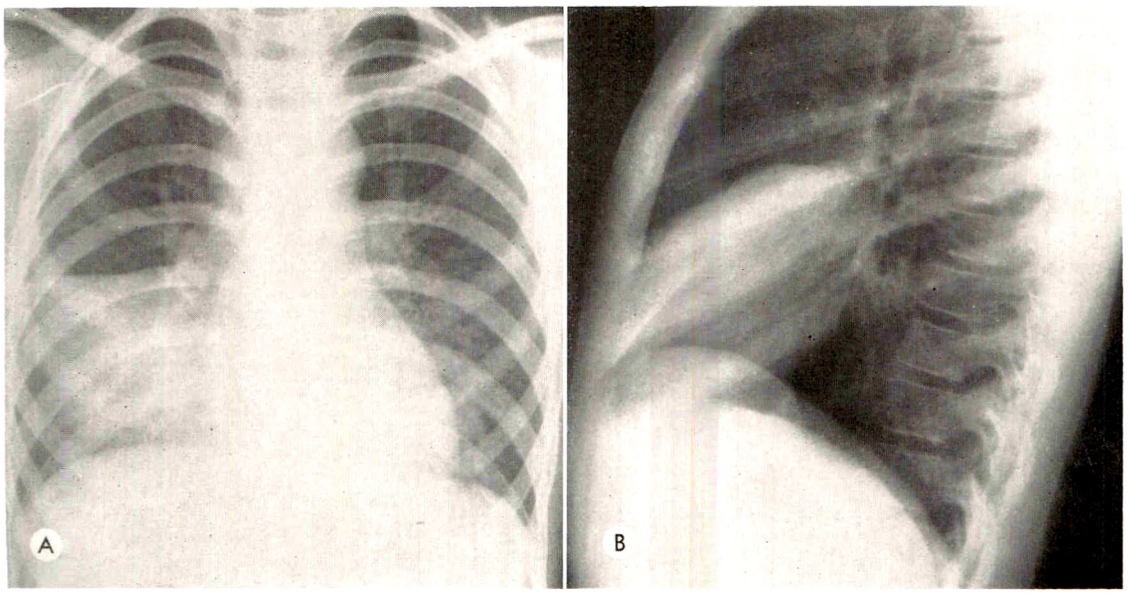


Fig. 6. Case II. (*A*) Posteroanterior and (*B*) lateral roentgenogram demonstrating atelectasis and pneumonia of the right middle lobe. Note the similarity to Case I (Fig. 1, *A* and *B*).

and anorexia and with roentgenograms disclosing recurrent infiltrates in the middle lobe and some degree of atelectasis. A bronchogram failed to demonstrate evidence of major bronchial obstruction. Bronchoscopy during an acute episode showed the right middle lobe bronchus to be completely occluded by inflammatory tissue.

Physical examination at this admission was essentially negative. A white blood cell count of 17,900 with a moderate left shift, and a hemoglobin of 13 gm. were reported. Bronchial washings showed no growth and the erythrocyte sedimentation rate was 41 mm./hr. Roentgen examination of the chest disclosed pneumonia with some atelectasis of the right middle lobe. Approximately one week later repeat roentgenograms showed some clearing.

Bronchoscopy immediately following admission revealed the right middle lobe orifice to be occluded by a red, round mass. Biopsy of this lesion was reported as hyperplasia of bronchial glands. Repeat bronchoscopy one week later showed no change and normal bronchial epithelium was obtained. Thoracotomy following the second bronchoscopic examination revealed a tumor mass in the right middle lobe bronchus that extended to the right main stem bronchus. A right middle lobectomy was performed and the pathologic report described a polypoid structure in the middle lobe bronchus arising from the wall and filling the lumen. Microscopic diagnosis was adenoma of the carcinoid type, subacute purulent bronchitis, obstructive bronchiectasis and chronic focal pneumonitis with organization.

Follow-up bronchoscopy six months later showed no evidence of recurrence. Chest roentgenograms at this time were not remarkable except for minimal pleural density in the right base. Five years after surgery the patient is without evidence of recurrent disease.

CASE III. J.A.B., a fourteen year old white female, was admitted to the Children's Hospital for evaluation of an intrathoracic tumor present since the age of eleven. Admissions during the preceding three years were associated with signs and symptoms of inflammatory disease of the chest. These consisted of fever, cough, left chest pain, wheezes and occasional sore throats. These symptoms improved following antibiotic and supportive therapy.

Physical examination at the time of the original admission revealed dullness to percussion and decreased breath sounds in the left base. There was also decreased vocal and tactile fremitus but no rales. On the second admission, there was dullness over the entire left chest with a few basilar rales. Physical findings of the current admission were essentially the same.

Initial roentgenograms showed patchy density in the left lower lobe (Fig. 7, *A* and *B*), which had only partially cleared at the time of discharge. As an outpatient, interval roentgenograms never showed complete clearing at the left base and fifteen months later the left chest was found to be totally opaque, probably

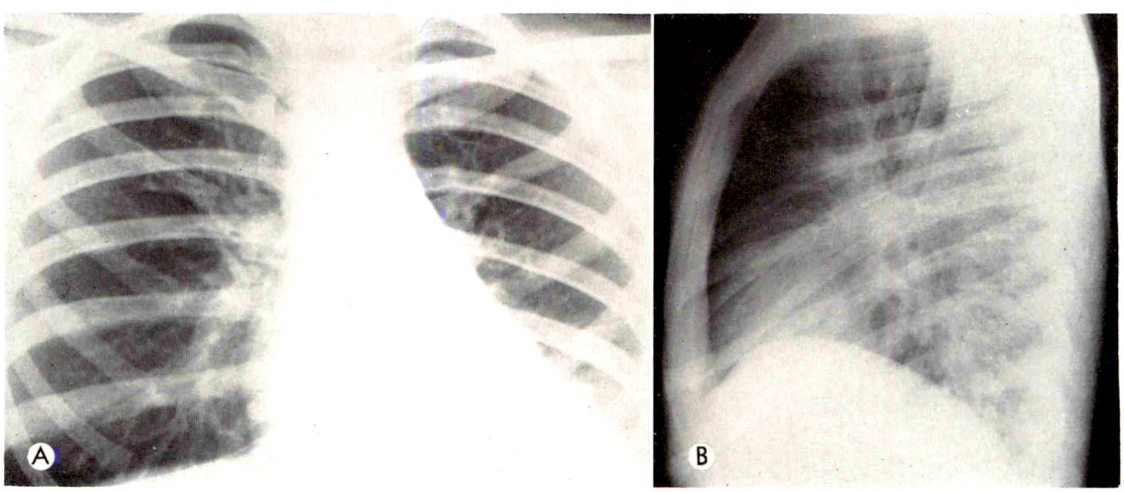


FIG. 7. Case III. (*A*) Posteroanterior and (*B*) lateral roentgenogram revealing patchy pneumonitis of the left lower lobe.

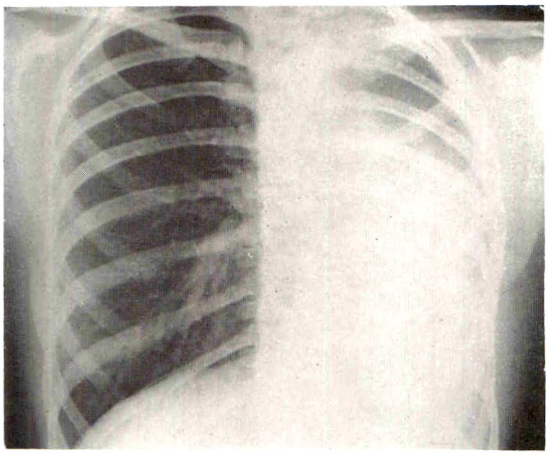


FIG. 8. Case III. Posteroanterior chest roentgenogram two months prior to surgery showing shift of the mediastinum to the left, secondary to atelectasis of the left lower lobe and pleural effusion.

due to pleural fluid. On the second admission there was a mediastinal shift toward the opaque side indicating at least partial collapse of the left lung. This condition persisted unchanged for the two years until the third admission (Fig. 8). Laboratory examinations during these admissions were not remarkable except for changes in the white blood cell count compatible with acute infectious diseases.

Bronchoscopic examination on the third admission revealed a firm fibrous mass in the left lower lobe bronchus. The pathology report was adenoma of the bronchus, with the diagnosis confirmed by several pathologists. Following biopsy and prior to definitive surgery, one episode of hemoptysis occurred. A pneumonectomy was performed approximately four years following the onset of symptoms. The pathology report on the surgical specimen revealed adenoma of the left lower lobe bronchus with extrabronchial extension, bronchiectasis and massive atelectasis.

A roentgen examination one year following surgery revealed no further change. Two years after surgery the patient was readmitted for complaints of a productive cough. Repeat bronchoscopy was negative and the patient was discharged. At the final admission (three years following definitive surgery and seven years following the onset of symptoms), bronchoscopy revealed nodules within the bronchial stump. A bronchopleural fistula developed and the patient expired.

An autopsy showed metastatic tumor to the pleura and the final diagnosis was bronchogenic carcinoma

TREATMENT

The treatment of bronchial adenoma has varied during the years; however, most methods of treatment have been confined to surgical extirpation, either bronchoscopically or by thoracotomy. Irradiation has been used by Foster-Carter,[18] Souders and Kingsley[59] and Naclerio and Langer[45] but generally has been abandoned, being ineffective with safe dosage.

Most of the authors[26,31] of earlier literature were of the opinion that bronchoscopic removal and fulguration was the treatment of choice. This may still be true in the older patient who is a poor surgical risk. Soutter[60] reported in 1954 that 20 of his patients were treated by bronchoscopic removal of the adenomas; these 20 patients averaged 15 bronchoscopic procedures apiece over a period of three and one-half years, and 1 patient had 75 different bronchoscopic procedures. Two of these patients died after the thirteenth and fourteenth procedures, respectively. Moreover, since 10 per cent of all bronchial adenomas have metastases, it becomes apparent that bronchoscopic removal cannot be considered an adequate operative procedure for cure of the tumor. Two-thirds of the 60 cases of Overholt *et al.*[47] had invasion of the bronchial wall.

Currently, open resection is considered the treatment of choice for the reasons listed above. The type of surgical procedure will depend upon the location and size of the tumor mass, accessibility of the regional lymph nodes, and the condition of the lung parenchyma distal to the tumor. The majority of cases will require either a pneumonectomy or a double lobectomy, but occasionally a single lobectomy may suffice. In rare instances of early, small lesions without chronic lung disease, bronchotomy with excision of the tumor might be possible.

The results of treatment are difficult to interpret because of the slow growing char-

acteristics of this tumor. At least ten year survivals are necessary for accurate evaluation. Overholt *et al.*[47] had a ten year follow-up on 20 patients. One of these succumbed to the tumor and 3 others had lymph node involvement at the time of death from other causes. This gives an 80 per cent ten year survival without known disease. Results of combined methods of treatment by Moersch and McDonald[43] reveal that 45 of 51 survived and 2 of the 6 deaths occurred more than ten years after treatment. All 21 patients reported by Knesevitch *et al.*[30] were alive and well at the time of their report, although 1 had a recurrence of the tumor; the duration of the follow-up was not stated. Soutter *et al.*[61] had an over-all five year survival rate of 73 per cent.

SUMMARY

The history of bronchial adenoma has been briefly described and the literature reviewed. Although scattered typical cases were reported in the nineteenth century, this disease has been recognized as an entity only since 1930. This potentially malignant neoplasm has been diagnosed with increasing frequency during the past three decades but, with the exception of the pediatric and otolaryngologic literature, little has been published concerning this entity in childhood.

We have summarized the literature describing this disease in children under the age of twelve and have added 3 previously unreported cases for a total of 19; 16 other cases were found to have been reported in youths between twelve and sixteen years of age.

Bronchial adenoma should be included in the differential diagnosis in children with a history of chronic cough, recurrent localized pneumonitis and atelectasis. A history of hemoptysis may be somewhat less common in childhood but increases the possibility of bronchial adenoma. Laminagraphy and bronchography are valuable assets in establishing the diagnosis. Bronchoscopy is then indicated.

With proper diagnosis the possibility of surgical cure is great.

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CONSIDERATIONS IN THE ROENTGEN DIAGNOSIS OF PNEUMONIA IN CHILDREN*

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ROENTGEN examination of a child's chest is, at best, an unpopular undertaking. Interpretation often varies in proportion to the number of radiologists offering opinions. The reporting of roentgen findings is, in many instances, attended by a lack of objectivity and skepticism about the possibility of accuracy. Our interest in this problem has led to the compilation and application of general principles governing roentgen technique, viewing, and interpretation. These principles are not original. They have been culled from the writings and teachings of many experienced radiologists and have been evaluated for an extended period. Adherence to these principles has enabled us to obtain a high percentage of chest roentgenograms of uniform quality, to approach the roentgenograms rationally, to utilize fairly reliable signs of focal areas of pneumonia, and to achieve general agreement in roentgen interpretation.

BACKGROUND

Kings County Hospital Center is a large general hospital with a proportionate number of pediatric beds. In a centralized radiology department almost all in-patient and emergency room services are performed. The pediatric emergency unit also functions as a twenty-four hour "open house" for out-patient services. It is the practice of the pediatric staff to order chest roentgenograms for virtually every child who is febrile or who has upper or lower respiratory symptoms. The volume of pediatric chest roentgenograms is, therefore, great, necessitating the attainment of maximum efficiency in radiologic services. During the winter months, between 40 and 50 pediatric chest roentgenograms in a 24 hour period are ordered on emergency room patients alone.

METHOD OF EXAMINATION

Uniformity in quality of chest roentgenograms is best achieved when one technician performs all pediatric examinations. In our institution, this is possible for only a limited period of the day. The next expedient is to insist on a procedure which can be performed most easily (and therefore most consistently) by all the technicians involved in pediatric work. Up to the age of 5 years, we rely entirely on a supine chest roentgenogram exposed at a 40 inch target-film distance at 1/60th of a second (1/120th of a second would be preferable). Immobilization is accomplished with sandbags, pinned towels, adhesive tape, or the technician's rapport with the child. The frequency of making exposures in full inspiration appears to be enhanced by placing the children in recumbent position.[5] The limitation to a single supine chest roentgenogram is time saving, is apparently adequate for satisfactory interpretation in most instances, and serves to emphasize to the technician the necessity for exposure of a single excellent roentgenogram. The roentgenogram is viewed immediately after automatic processing to determine the necessity for repeat examination or additional views.

ROENTGENOGRAM EVALUATION

Our approach begins with an evaluation of the quality of the roentgenogram. We purposely direct our attention to this consideration before attempting interpretation. The presumption is maintained that

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the roentgenogram should be comparable in quality to an adult chest roentgenogram for our accurate interpretation. Albeit the attainment of equal quality is considerably more difficult, we feel that compromise gives license to the technician to relax his efforts, and, in addition, precludes accurate roentgen diagnosis. This attitude is troublesome for everyone at the outset, but insistence on good quality pediatric chest roentgenograms has proved fruitful for us.

We test the quality of the roentgenogram with four considerations:

1. Is the roentgenogram properly exposed? For our purpose, we feel that the roentgenogram is adequately exposed if the thoracic vertebrae can easily be identified through the cardiac density.

2. Is the child properly centered? Needless to say, the central ray should transect the midthorax (a roentgenogram exposed for "chest and abdomen" is unsuitable for accurate evaluation of the chest). The presence or absence of rotation is most easily determined by examining the relative lengths and symmetry of the clavicles (Fig. 1*A*). The position of the trachea is a less reliable sign for several reasons. Turning the child's head can alter the position of the trachea in an otherwise well-centered roentgenogram. Buckling of the trachea due to incomplete inspiration can simulate rotation. On the other hand, the trachea may appear to be in the midline, while the clavicles give evidence of rotation. Slight degrees of rotation result in differences in the two sides of the thorax, due to differences in amounts of soft tissue and aerated lung traversed by the roentgen rays.[1] Minor rotational differences are often mistaken for unilateral pneumonia by the unwary interpreter.

3. Is the roentgenogram exposed in inspiration? In the absence of clinical evidence of heart disease, the adequacy of inspiration can be assessed by determining the relationship of the cardiac apex to the left dome of the diaphragm. In full inspiration, the cardiac apex is well above the diaphragm (Fig. 2, *A–C*). In addition, if the head is not turned, the tracheal air shadow will be vertical and straight. Counting the number of ribs above the dome of the diaphragm is not necessary; and, if the central roentgen ray is too high or too low, a misleading impression may be obtained. This can easily be appreciated by examining the slope of the diaphragm in the lateral

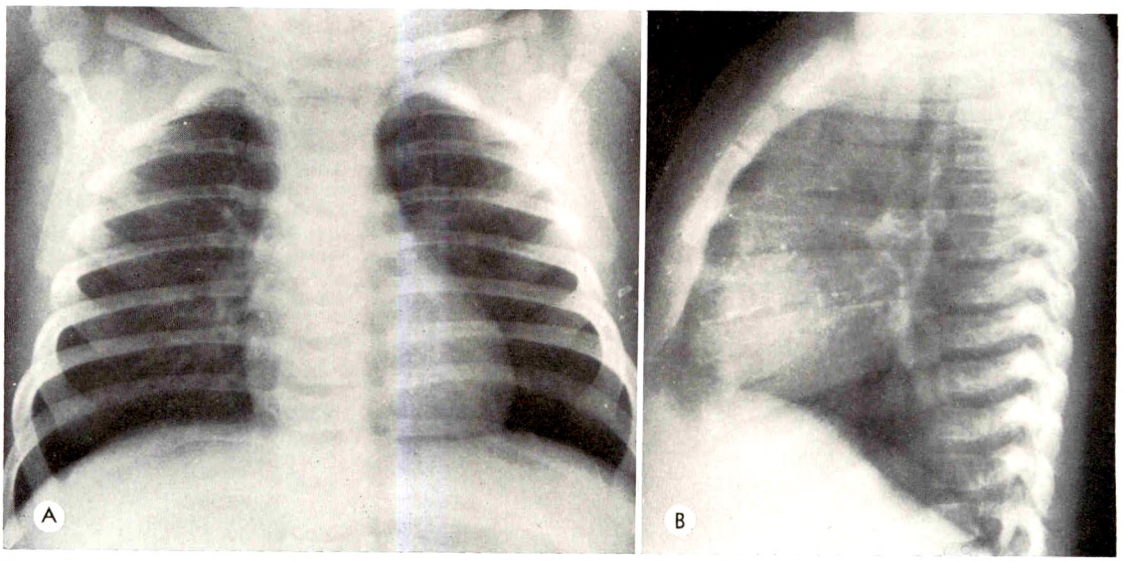


FIG. 1. Normal chest. (*A*) Anteroposterior view. Note the symmetry and equal lengths of the clavicles indicating proper centering. (*B*) Lateral view. Note caudal sloping of the diaphragmatic domes dorsally.

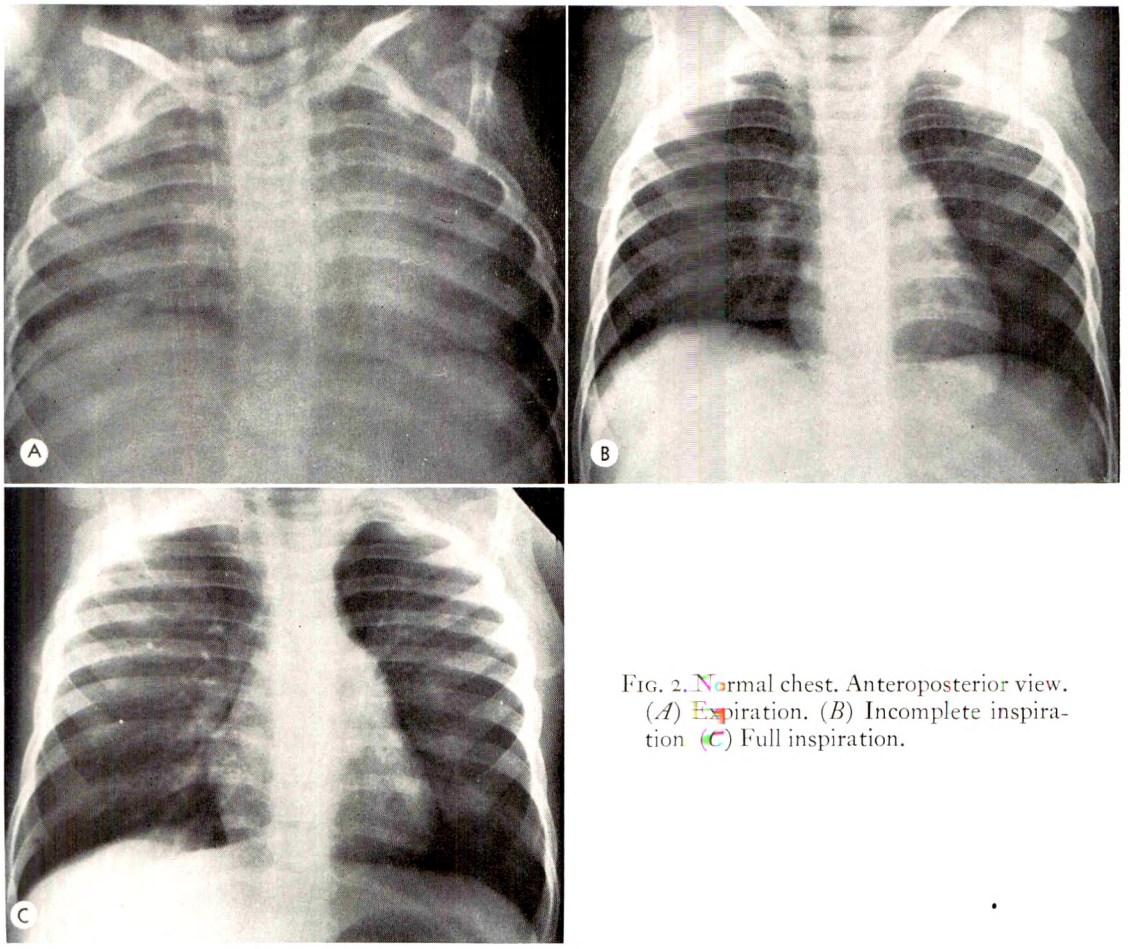


Fig. 2. Normal chest. Anteroposterior view. (*A*) Expiration. (*B*) Incomplete inspiration. (*C*) Full inspiration.

position (Fig. 1*B*). The diaphragm is seen to slope caudad dorsally. Therefore, if the central ray is too high, the diaphragm will be projected caudad on the roentgenogram, simulating full inspiration. If the central ray is too low, the diaphragm will be projected cephalad. Occasionally, when exposing an upright chest roentgenogram, the thorax will appear foreshortened due to the child's leaning forward against the cassette holder.

4. Is motion absent from the roentgenogram? The domes of the diaphragm should appear razor-sharp if no respiratory motion or movement mars the roentgen exposure.

If these questions can be answered in the affirmative, we consider the roentgenogram to be adequate for critical evaluation. A simple cardboard rectangle with the projected cone size cut out, mounted on the shadow box to exclude extraneous light, has enabled us to view the chest roentgenogram with greater facility. The use of a cardboard cylinder from which paper toweling has been removed, placed against the "good eye," is recommended for viewing localized portions of the lung fields. These preliminary measures may seem rather elaborate, but serve to emphasize the care with which the child's chest roentgenogram should be approached if iatrogenic pneumonias are to be avoided.

Increased Bronchovascular Markings

We feel that the roentgen diagnosis of "increased bronchovascular markings" should be condemned. The inference evoked by this diagnosis is that the viewer is

thoroughly acquainted with the normal variation in size of vessels. Caffey[1] citing observations by Maresh and Washburn, states that the size of hilar markings is subject to wide individual variation in normals and bears no relationship to the severity or frequency of respiratory infections. Too often, rendering a diagnosis of "increased bronchovascular markings" is an act without conviction, supporting almost any clinical diagnosis under consideration. Under these circumstances, it represents an evasion for the radiologist, a *carte blanche* for the clinician's imagination, and a disservice to the patient. The expression, "increased bronchovascular markings" has so many meanings that it has become meaningless to us.

Careful attention to the characteristics of normal vessels will enable the viewer to identify alterations and to relate them to specific pathologic disorders. Normally, the vessels course in a linear fashion, taper gradually, are sharply marginated and clearly branching. One or two vessels are usually seen *en face*, adjacent to each hilus. In congenital cardiac disorders with increased pulmonary blood flow, the abnormal volume of blood in the lung distends the arteries and veins, resulting in the roentgen appearance of tortuous vessels, crowded together near the hili so that their margins are poorly differentiated (Fig. 3*A*). The increased pulmonary blood flow can often be appreciated by observing that the number of vessels seen *en face* near the hilus is increased to four or more and that an occasional large vessel is seen *en face* at an inordinate distance from the hilus (Fig. 3 *B*). Often, the size, number, and distribution of vessels *en face*, resulting from increased pulmonary blood flow, is appreciated more easily than the character of vessels seen in profile. In addition to congenital cardiac disorders with systemic-pulmonary shunts, an increased volume of blood occurs in the lung in severe anemia, glomerulonephritis, and left ventricular failure. We find that, in the latter instance, the increased volume of blood in the lung is not commonly appreciated roentgenographically. In the presence of systemic-pulmonary shunts, the presence of associated congestive failure or bronchopneumonia cannot be determined with certainty.

Alterations in the appearance of normal vessels also occur in children with fibrocystic disease of the lung and with bronchial asthma. Recurrent episodes of bronchopneumonia cause chronic inflammatory thickenings. The proximity of the bronchi to pulmonary vessels results in irregular and unsharp margination of the vessels roentgenographically. The nature of the

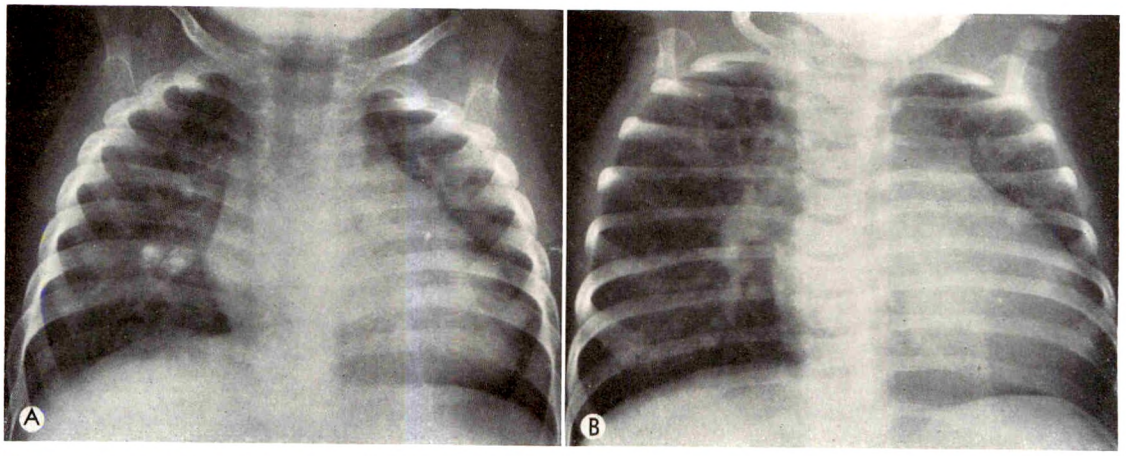


Fig. 3. Congenital heart disease with systemic-pulmonary shunt. (*A*) Note the enlarged, tortuous, poorly marginated vessels at the hili. (*B*) The presence of increased pulmonary blood volume is manifest by the number of vessels *en face* in the middle third of the right lung field.

pathologic process can be determined when thickened bronchial walls are seen *en face* clustered about the hili (Fig. 4), or in profile at the lung bases.[4] Alterations in pulmonary blood volume secondary to cardiac disorders and alterations in bronchovascular outline due to chronic inflammatory thickenings are the only roentgen changes that we can consistently recognize.

THE CHEST ROENTGENOGRAM OF A NORMAL CHILD

Examination of the chest roentgenogram begins and lingers with a critical evaluation of the quality of the study under proper viewing conditions. The right dome of the diaphragm is easily seen from the lateral costal margin to the cardiophrenic angle. The left dome of the diaphragm is normally seen throughout the lateral two-thirds, and, occasionally, the lateral three-quarters of its extent. In an adequately exposed roentgenogram, the margin of the diaphragm is evident, even when projected over a rib. Only that segment of diaphragm, which exactly coincides with cortical rib margin, is obscured. The cardiomediastinal margin is seen along its entire length, except for interruption at the hili by the densities of neighboring vessels. Rarely, the lower cardiac margin is interrupted by an adjacent vessel coursing obliquely.[3] The margin of a normal left atrium can be seen occasionally to the right of the midthoracic vertebrae. One or both margins of the horizontal fissure are usually evident. The vertical fissure,[2] often detected on chest roentgenograms of children without heart disease, is most often seen on the right, occasionally on the left or bilaterally. The vertical fissure is described by Davis[2] as the lateral portion of the major fissure which has been directed in an anterior position, so that it is in a sagittal plane. The fissures are normally razor-sharp and razor-thin. The lateral margins of the costal pleura hug the inner cortical rib borders in the caudad two-thirds of the thorax. The densities of the subcostal muscles[1] occupy the lateral

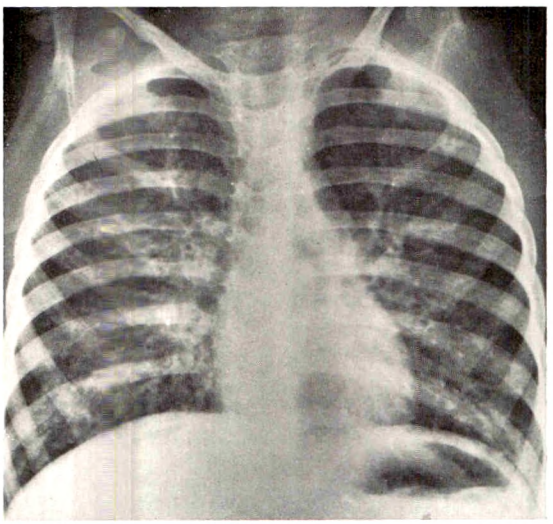


Fig. 4. Fibrocystic disease of the lung. Thickened bronchial walls are seen *en face* at the hili.

margins of the first three interspaces. The lateral lung margin bulges slightly in the interspaces. This convexity may be exaggerated on a normal chest roentgenogram (simulating overdistended lungs) if the central ray was caudad, relative to midthorax. When the chest roentgenogram is adequately exposed, air is normally seen in the main stem bronchi, frequently in the segmental bronchi behind the heart, and occasionally in the bronchi in the medial third of both lung fields.

SPECIFIC ROENTGEN SIGNS OF PNEUMONIA

Our purpose in studying large numbers of chest roentgenograms was twofold: to learn and relate the limits of normal variation and to indicate how normal limits are exceeded in terms of specific roentgen signs. Adoption of these specific roentgen signs by other members of our department has enabled us to constantly test their applicability and validity. Three signs have proved useful. Four signs will be described:

A. *Obliteration Sign.* This is our designation for the silhouette sign, described and popularized by Felson.[3] Any segment of the diaphragmatic or cardiomediastinal margin (see exceptions, above) which is not clearly defined is obliterated by the presence of

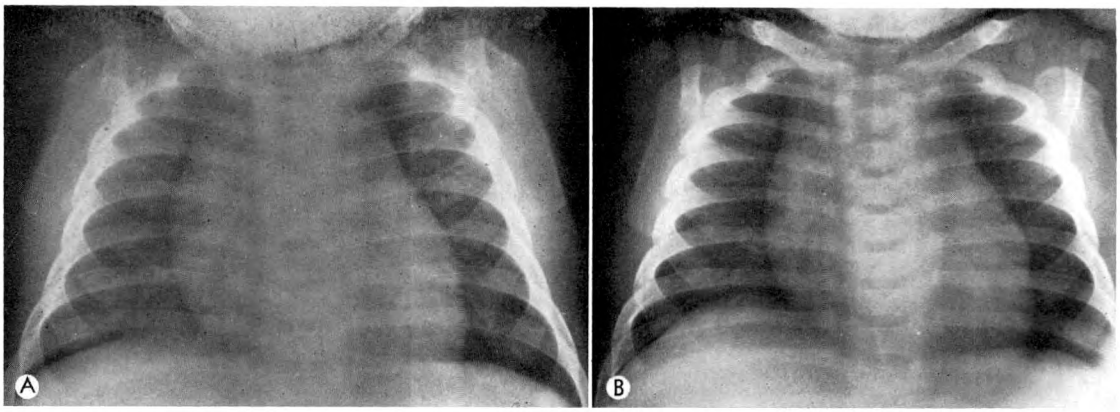
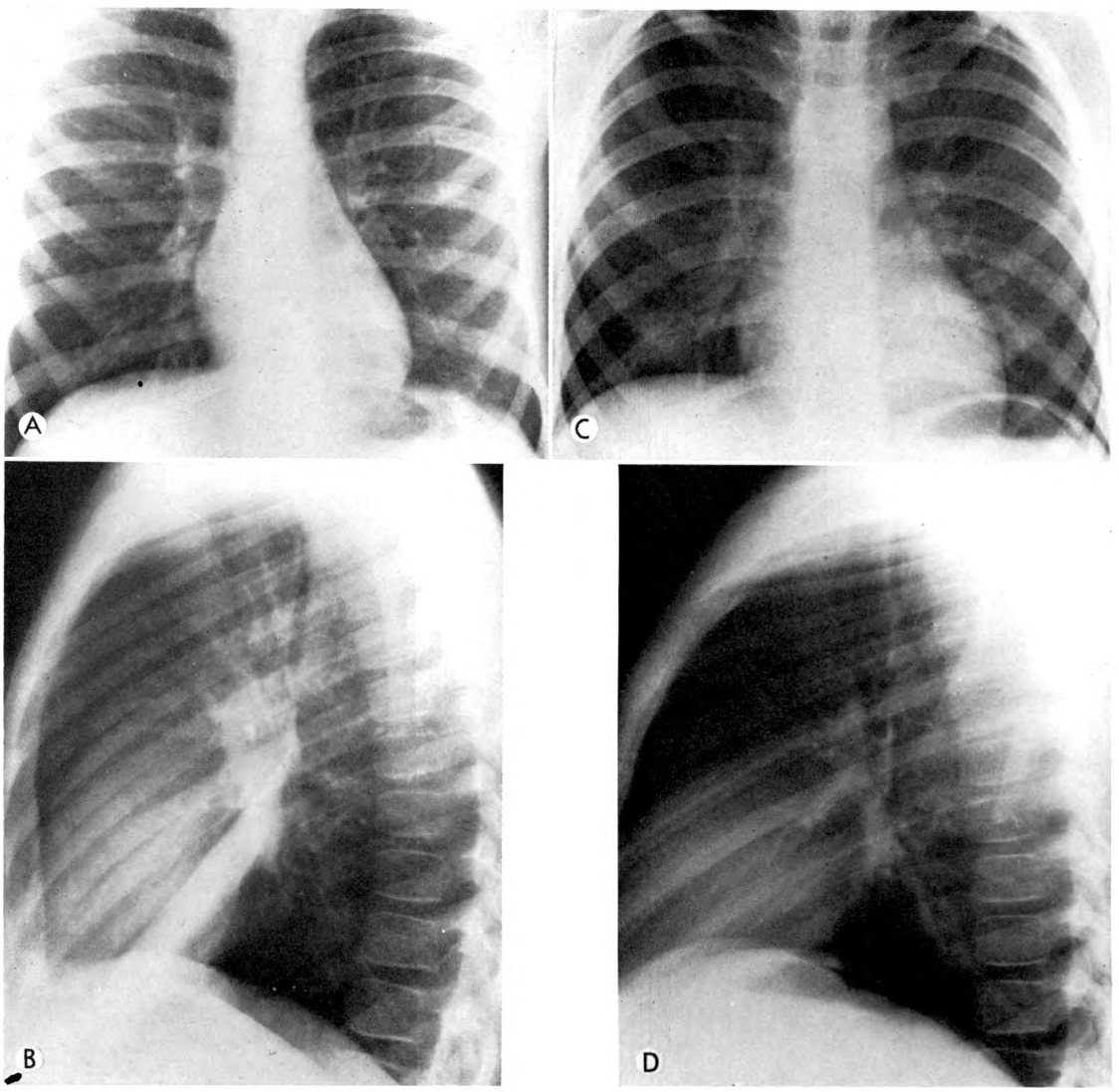


FIG. 5. Obliteration sign. (*A*) The medial third of the right dome of the diaphragm is obscured by pneumonia in the right lower lobe. (*B*) The right dome of the diaphragm is easily seen after the pneumonia has cleared.



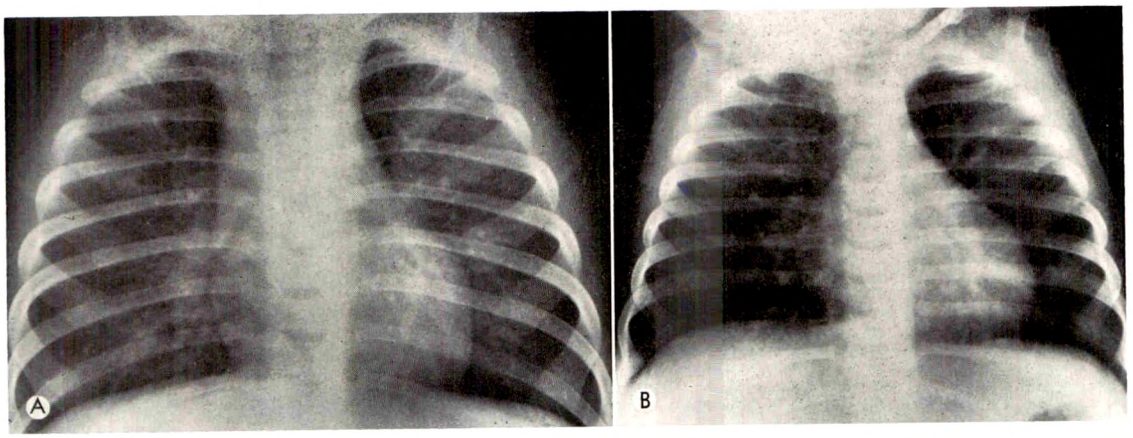


Fig. 7. Air bronchogram sign. (*A*) Right middle lobe pneumonia of medial segment. Air streaks in a portion of lung in which vascular outlines are obliterated. (*B*) Pneumonia of posterior segment of right upper lobe.

parenchymal (or pleural) densities (Fig. 5, *A* and *B;* and 6, *A–D*).

B. *Air Bronchogram Sign.* This is essentially the air bronchogram sign of Felson.[3] Air in branching bronchi is clearly visible in the presence of peribronchial infiltrates or alveolar consolidation. These pathologic processes, by necessity, obscure or obliterate the margins of adjacent vessels. The association of these two components constitutes the *air bronchogram* sign. The resultant roentgen finding of black branches on a white background can easily elude the viewer if not specifically sought for (Fig. 7, *A* and *B*).

C. *Retrocardiac Sign.* This is the air bronchogram sign applied to the medial segment of the left lower lobe. Normally, branching bronchi *and* branching vessels can be identified behind an inherent cardiac density. Retrocardiac pneumonia will obliterate the vascular margins and increase the apparent cardiac density (Fig. 8, *A* and *B*).

D. *Shaggy Vessel Sign.* We attempted to identify focal areas of parenchymal infiltration by examining minutely the characteristics of regional vessels and detecting alterations in outline, tapering, and branching. Occasionally, we are able to assure ourselves, after examining a roentgenogram of good technical quality, that a segment of lung is traversed by "chunky" vessels, signifying focal infiltration. We are often able to substantiate this finding by a change in the appearance of these vessels on a subsequent roentgenogram of comparable quality. However, we find this type of judgment to be of limited usefulness because of our own inconsistency and because of the frequent disagreement among other viewers. We, therefore, feel that the diagnosis of "increased bronchovascular markings" by a viewer who fails to critically analyze roentgenogram quality and who reviews a limited number of normal studies is guesswork.

Our reluctance to employ this sign implies that we fail to diagnose "all" focal areas of pneumonia *per primum*. This failing is not restricted to the child's chest roentgenogram. Occasionally, we are un-

Fig. 6. Obliteration sign. (*A*) The left dome of the diaphragm appears indistinct. (*B*) Lateral view. Consolidation in the anteromedial basilar segment of the left lower lobe, responsible for the diaphragmatic finding, is best seen on this view. (*C*) The diaphragm is clearly visualized after resolution of the pneumonia. (*D*) Lateral view.

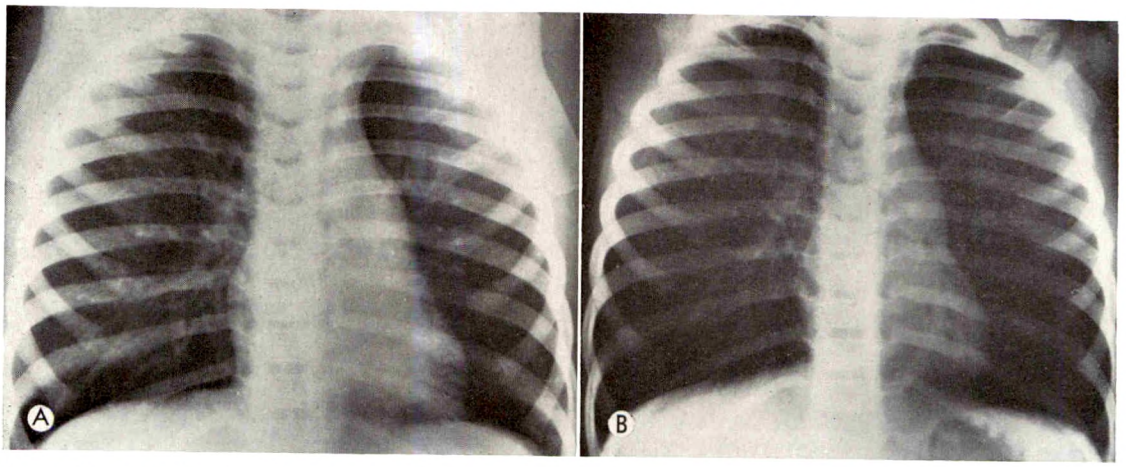


FIG. 8. Retrocardiac sign. (*A*) Pneumonia of left lower lobe, posteriorly. Vascular outlines are obliterated. Increased soft tissue density is present medially. (*B*) After resolution, vascular margins are evident. "Cardiac density" has returned to normal.

able to detect marginal abnormalities in adult chest roentgenograms until we study previous and subsequent examinations for comparison. The diagnosis of pneumonia can often be made by the experienced clinician in the absence of detectable roentgen abnormality. However, when abnormal physical findings are not present, reliance on an accurate roentgenographic interpretation becomes important. The roentgen diagnosis can be established with reasonable certainty if the limitations of the roentgenologic method are respected. The chest roentgenogram becomes valueless if the viewer fails to discriminate between normal and pathologic findings. The abuse of the roentgenologic method, by failing to establish the roentgen criteria of normality, may defeat efforts to establish an accurate clinical diagnosis and may delay proper therapy.

ROENTGEN SIGNS OF A CHILD IN RESPIRATORY DISTRESS

Mention has been made of bulging intercostal spaces as a sign of overinflation on a roentgenogram which is properly centered. It is often difficult to decide between full inspiration and overinflation in a chest roentgenogram in which the diaphragm appears depressed, flattened, and obliquely pitched. Clinical examination of the child,

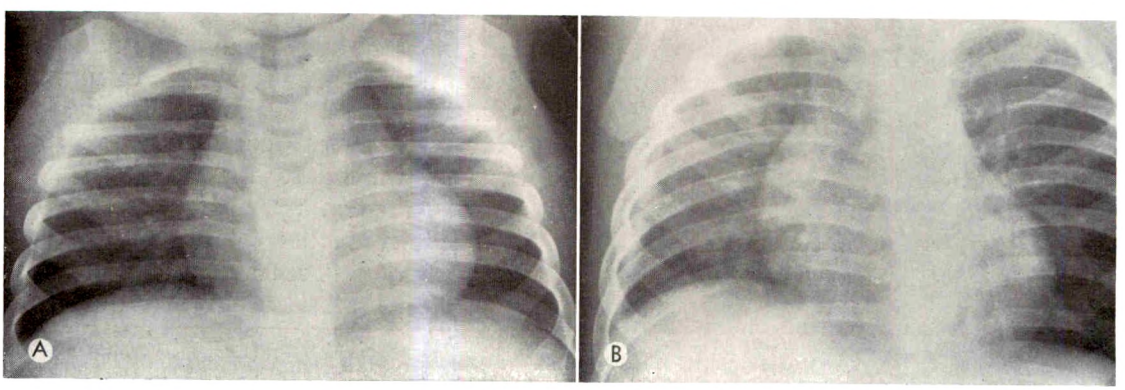


FIG. 9. (*A*) Anteroposterior view. Vertical fissure, on right, is thickened and poorly marginated laterally. (*B*) Right lateral decubitus view. Pleural exudate, previously noted in the vertical fissure, has gravitated to the right lateral costal margin. Pneumonia was subsequently detected.

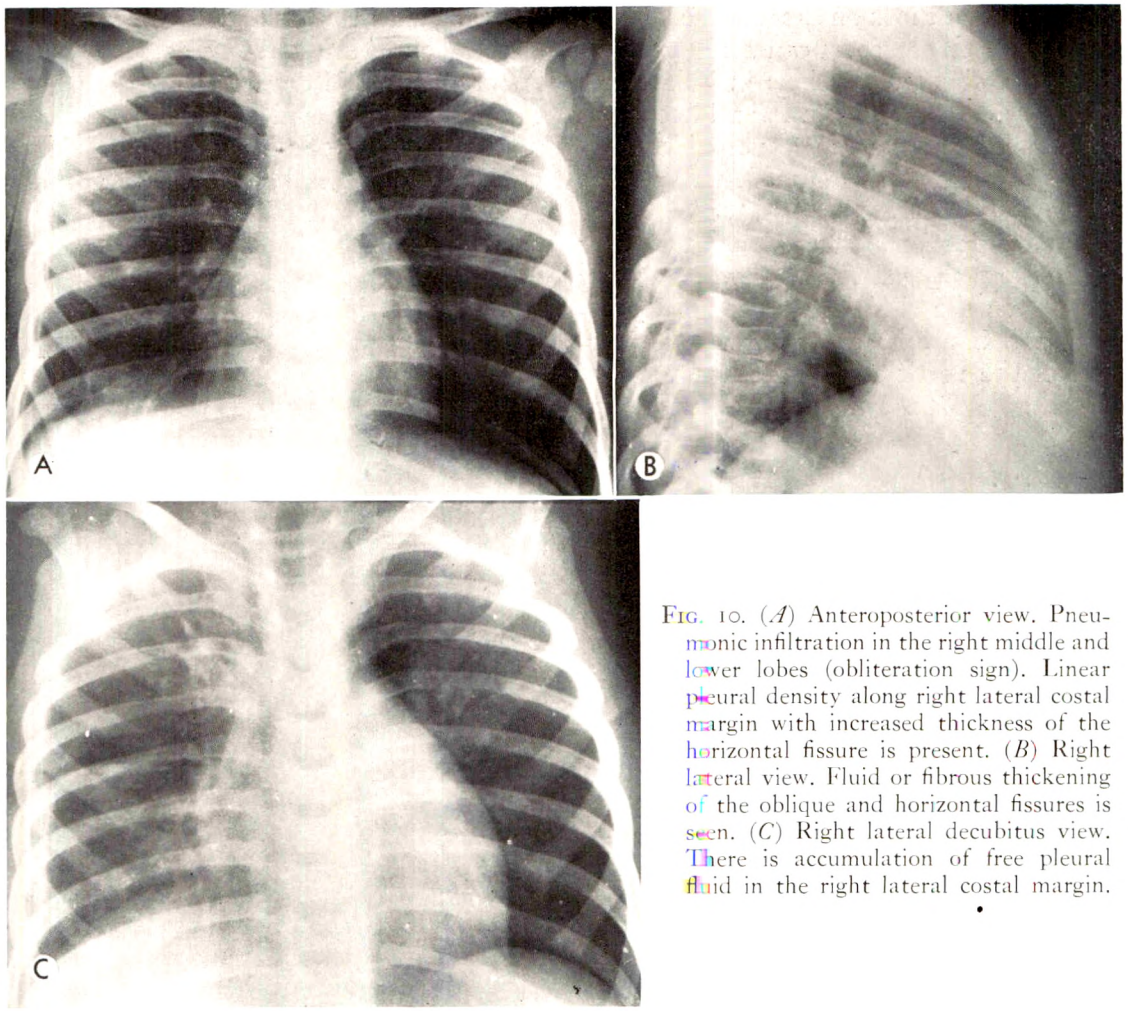


Fig. 10. (*A*) Anteroposterior view. Pneumonic infiltration in the right middle and lower lobes (obliteration sign). Linear pleural density along right lateral costal margin with increased thickness of the horizontal fissure is present. (*B*) Right lateral view. Fluid or fibrous thickening of the oblique and horizontal fissures is seen. (*C*) Right lateral decubitus view. There is accumulation of free pleural fluid in the right lateral costal margin.

chest fluoroscopy, repetition of this finding on a second roentgenogram, or demonstration of an abnormal retrosternal clear space on a lateral view—all can aid in differentiation.

Compelling clinical evidence of respiratory distress may warrant further roentgen examination of a child whose initial chest roentgenogram appears normal. We may re-order a supine chest roentgenogram im-

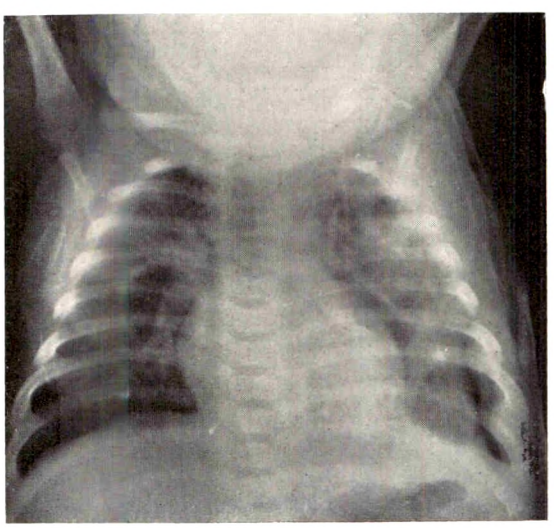


Fig. 11. Staphylococcal pneumonia. Radiolucencies, probably representing loculated pyopneumothorax (on left), ? abscesses, ? foci of obstructive emphysema, ? irregular bands of pleural adhesions.

mediately as a check on the initial examination. Chest fluoroscopy can often give information about alterations in pulmonary dynamics. We feel that a lateral decubitus chest roentgenogram is a valuable diagnostic adjunct in this situation. We have occasionally detected small amounts of pleural exudate on a lateral decubitus study before parenchymal densities were evident and before the clinicial syndrome was established. Careful attention to the appearance of the vertical fissure, when present, may enable detection of interlobar pleural exudates (Fig. 9, *A* and *B*). A repeat supine chest roentgenogram within several hours may demonstrate the subsequent appearance of parenchymal densities. The rapidity of onset of serious illness in a child warrants close cooperation between the clinician and the radiologist in these instances.

SOFT TISSUE COMPANIONS AND SIMULANTS OF PNEUMONIA

The demonstration of a soft tissue density having the appearance of pneumonia necessitates appreciation of other soft tissue densities which mimic or accompany pneumonia. Enlarged lymph nodes are said to be present commonly in lower respiratory infections[1] and can be indistinguishable from segmental consolidations. Small focal areas of atelectasis also mimic and accompany pneumonic infiltrates. Lobular atelectasis can be identified by its oblique linear streaking. Segmental and lobar atelectasis are recognized by characteristic patterns of orientation on recumbent and lateral chest roentgenograms. Pleural transudates and exudates, because of their ubiquitous manifestations as loculations adjacent to costal, mediastinal, interlobar, perilobar, and supradiaphragmatic surfaces,[1] cause the most perplexing problems in roentgen differentiation.

It is important to appreciate the frequency with which pleural exudate accompanies segmental or lobar consolidation. Fluid may accumulate in the adjacent interlobar spaces or in the lateral costal margin. The importance of detecting pleural fluid in any child with respiratory distress has been already emphasized. A linear soft tissue density along the lower costal margin should alert the radiologist to the presence of pleural fluid or pleural thickening. A lateral decubitus chest roentgenogram will confirm the presence of free pleural exudate and will enable the clinician to make a specific bacteriologic diagnosis and institute immediate appropriate therapy (Fig. 10, *A–C*).

RADIOLUCENT COMPANIONS OF PNEUMONIA

Necrotic foci may be present in areas of parenchymal consolidation. These foci remain as soft tissue densities indistinguishable from surrounding consolidation until they communicate with a bronchus and replace their contents with air. The resultant radiolucencies can easily be confused with focal areas of obstructive emphysema, foci of re-aerated lung in the midst of consolidation, loculated pyopneumothorax, and the mosaic of irregular pleural adhesions (Fig. 11). Differentiation is difficult, even with review of serial chest roentgenograms. Re-aerating lung becomes rapidly evident, while cavitation, obstructive emphysema, and pleural radiolucencies may persist for variable periods of time. Eventual regression of these processes is the rule.

SUMMARY

An accurate roentgen diagnosis of pneumonia in childhood necessitates a critical evaluation of the quality of the roentgenogram. Assessment of proper exposure, centering, degree of inspiration, and absence of motion is made before interpretation is undertaken. Observations pertaining to the normal chest roentgenogram in childhood are outlined. Avoidance of the expression, "increased bronchovascular markings" is emphasized. Specific alterations in the normal vascular structures may be recognized and may be related to pathologic disorders responsible for their occurrence. Focal areas of pneumonia are identified by utilization of three specific signs:

the obliteration, the air bronchogram, and the retrocardiac density signs. The soft tissue densities which mimic and accompany pneumonia as well as the radiolucencies commonly present are detailed. A program for the roentgen evaluation of a child in acute respiratory distress is proposed.

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The author is grateful to Drs. John Caffey, Robert Ledner, Hooshang Taybi, and Carl Scheer for many ideas expressed in this paper.

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ANTENATAL ROENTGEN DIAGNOSIS OF MECONIUM PERITONITIS*

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MECONIUM peritonitis is a chemical peritonitis which is produced by the passage of meconium from the fetal intestinal tract into the peritoneal cavity. In the majority of cases the communication has closed by the time of birth. Intestinal obstruction may or may not be present.

The areas of chemical peritonitis frequently serve as sites for *in utero* calcium deposition. The importance of the roentgen detection of these irregular, amorphous appearing areas of calcification in making a diagnosis of meconium peritonitis in the newborn infant was emphasized in 1944 by Neuhauser.[3] The only published case of antenatal roentgen diagnosis of meconium peritonitis was recorded in 1956 by Harris and his associates.[2] Calcium deposits had been identified in the area of the fetal abdomen on maternal pelvimetry films. It has seemed desirable to record a second case in which an antenatal diagnosis of meconium peritonitis was made from the observation of calcium deposits in the area of the fetal abdominal parts on pelvimetry roentgen studies.

REPORT OF A CASE

The patient, a twenty-one year old para 0, gravida 1, white married woman, was admitted to the Ball Memorial Hospital on November 29, 1960 because of the recent onset of relatively severe labor pains. The duration of her pregnancy had been thirty-seven weeks by calculation. The pregnancy had been normal except for the occurrence of rather marked abdominal distention during the last month of pregnancy. An upper respiratory infection had been treated satisfactorily with penicillin during the third month of pregnancy. The pregnancy had not been associated with vomiting, bleeding, albuminuria or hypertension. A diagnosis of probable polyhydramnios with overdistended uterus was made prior to the onset of labor. Her total weight gain had been 21 pounds. She had gained 7 pounds in the last month of pregnancy. This weight gain and the significant increase in abdominal enlargement had not been controlled by the use of dietary restrictions and diuretics. There had been no symptoms of toxemia. Abdominal palpation had revealed a single intrauterine pregnancy with a normal sized infant. Spontaneous labor had occurred at thirty-seven weeks of gestation.

The patient's past history was essentially negative. She had experienced an episode of rheumatic fever at twelve years of age. There had been no residual heart disease. She had never been operated upon. There was no significant family history of illness or congenital malformation. Her urine examination on admission to the hospital was within normal limits. Her hemoglobin was 9.8 gm. and her hematocrit was 30 per cent. The white blood cell count was 11,200 with a normal differential. The patient was Rh positive and her serology was negative.

At the onset of labor, the presenting part, the head, was unengaged. After approximately twelve hours of active labor, a sterile vaginal examination was carried out and the membranes were ruptured mechanically with the escape of over 2,000 cc. of clear amniotic fluid. Roentgenographic pelvimetry was requested after twenty-four hours of labor because of polyhydramnios, failure of satisfactory progress of labor and failure of descent of the fetal head. The presence of fetal congenital malformations was suspected. In addition, the possibility of a cephalopelvic disproportion was considered.

The pelvimetry films showed a single uterine pregnancy in cephalic presentation. The fetal head was at the inlet but was not engaged. There was no evidence of cephalopelvic disproportion. The placental position appeared to

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be posterior and superior. The fetal abdomen appeared large and the subcutaneous fat line of the anterior abdominal wall was poorly delineated. In addition, numerous amorphous, ill-defined calcium deposits were identified in the soft tissues of the abdominal wall of the fetus. These calcium deposits were visualized best in the lateral view (Fig. 1). Because of the occurrence and anatomic location of these calcium deposits, an antenatal diagnosis of meconium peritonitis was made.

After forty-eight hours of labor, delivery was carried out by the use of low forceps and an episiotomy. The head was delivered with ease. Marked dystocia, however, was encountered with delivery of the fetal body due to massive abdominal distention. Considerable extension of the episiotomy was required for delivery of this enlarged abdomen. The infant breathed and cried.

The infant weighed 7 pounds and 9 ounces at birth. There was marked abdominal and scrotal enlargement (Fig. 2). A diagnostic paracentesis

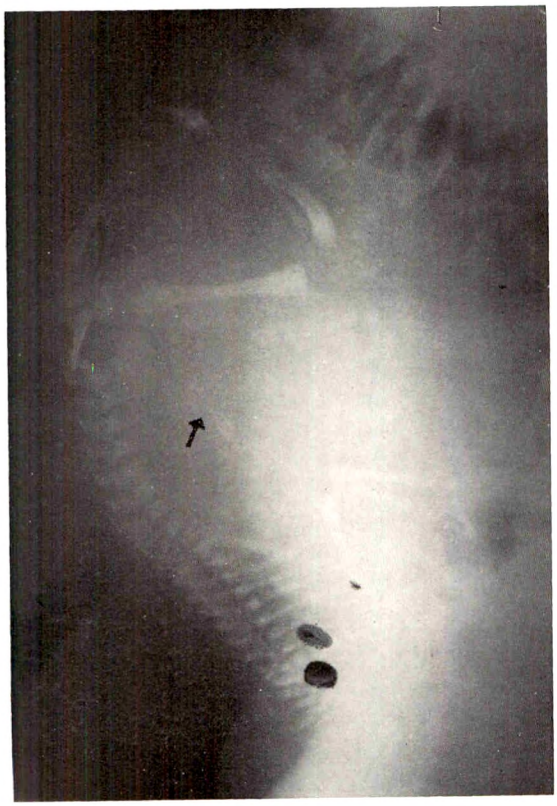


FIG. 1. Lateral pelvimetry film of the abdomen showing enlarged fetal abdomen with areas of fetal abdominal calcification.



FIG. 2. Photograph of the newborn male infant made immediately after birth showing abdominal and scrotal enlargement.

was performed shortly after birth. Approximately 500 cc. of brownish meconium liquid containing flecks of vernix caseosa was removed. A considerable amount of fluid remained in the enlarged peritoneal cavity and an additional 500 cc. of fluid was removed a few minutes later at operation. The presence of meconium peritonitis with calcium deposits around the peritoneal cavity was confirmed at operation. The patient was operated upon two hours after delivery. The peritonitis was caused by an antenatal ileal perforation. An extensive small bowel resection was carried out successfully. An additional operation, gastrojejunostomy, was required for relief of duodenal obstruction at fourteen days of age. The presence of calcium deposition in the peritoneal cavity and in the enlarged scrotum was demonstrated on post-

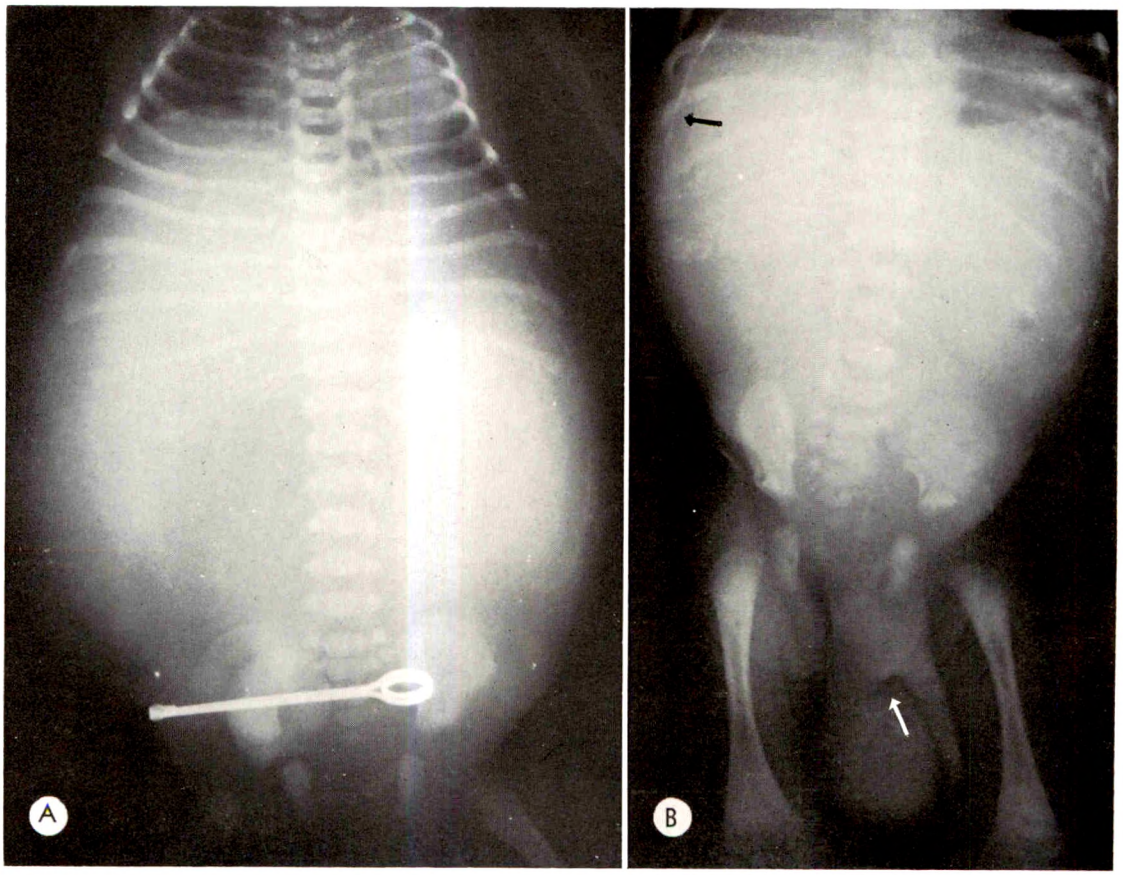


FIG. 3. Roentgenograms showing the appearance of the distended abdomen at birth (*A*) and the appearance at one month of age (*B*). The calcification is obscured on *A* due to the massive intraperitoneal fluid accumulation. Intraperitoneal and scrotal calcium deposits are more apparent on *B*.

natal roentgenograms of the infant (Fig. 3). Microscopic study of portions of the resected small intestine revealed the presence of calcium deposits in areas of organizing serosal exudate. The infant did well following the last operation and was eating and gaining weight satisfactorily at the time of his discharge from the hospital at forty-two days of age. He weighed five pounds and eight ounces at the time of his release from the hospital. He has continued to grow and develop normally during the three and a half months since his discharge.

DISCUSSION

Meconium peritonitis of the newborn indicates perforation of the bowel occurring in prenatal life or shortly after birth. Frequently, the site of perforation will seal over *in utero* and the infant will have no significant clinical findings at birth. In those cases in which the perforation persists, the postoperative mortality rate is very high. The most common cause of perforation is due to intestinal obstruction secondary to stenosis or atresia of the bowel. Other causes reported are intussusception, volvulus, peritoneal bands, Meckel's diverticulum, imperforate anus, meconium ileus and trauma.

The significance of intraabdominal calcification in the roentgen diagnosis of meconium peritonitis was reported by Neuhauser.[3] Calcification with meconium peritonitis has been reported as early as six months' gestation. At birth, the main peritoneal cavity and the processus vaginalis to the scrotum freely communicate, thus accounting for the scrotal swelling and peritesticular calcification.

The case reported by Harris and his associates[2] was a thirty-three year old para II who experienced marked abdominal enlargement during the eighth month of pregnancy. A clinical diagnosis of polyhydramnios had been made. Pelvimetry films showed excessive intrauterine fluid. In addition, the fetal abdomen appeared larger than normal. On the lateral films, a large number of amorphous, small calcific density deposits was seen in the area of the fetal abdominal wall. A diagnosis of meconium peritonitis was made. Labor was induced and a 5 pound and 13 ounce female infant was delivered. The presence of the calcium deposits in the area of the fetal abdomen was confirmed on postnatal films of the infant. The meconium peritonitis was associated with an ileal atresia. The infant was operated upon but did not recover.

In the case reported by Westdahl and Cline[5] in 1956, an oval ring of calcific density was observed in the area of the fetal abdomen on maternal roentgenograms taken at eight months of gestation. The finding was interpreted as a calcified fibroid tumor of the uterus. The roentgenograms were taken to evaluate the status of fetal age approximately two months after the mother had been involved in a severe automobile accident. The infant was found to have a palpable abdominal mass at birth. Abdominal roentgenograms showed the mass to be the calcific oval ring observed on the antenatal roentgenograms. The patient was asymptomatic until intestinal obstruction occurred at eleven weeks of age. The infant was operated upon and the mass was found to be a calcified meconium cyst, about the size of a small chicken egg, with which a loop of jejunum communicated. The mass was resected and the hole in the jejunum was closed. The intestinal obstruction was relieved and the infant recovered.

The only other case of meconium peritonitis in which antenatal findings have been recorded was reported by Ezes and his associates[1] in 1956. Their patient was studied because of polyhydramnios and marked abdominal enlargement during the last trimester of pregnancy. An abdominal puncture was carried out and 1,500 cc. of fluid was removed. Sixty cubic centimeters of radiopaque material were injected through the needle (amniography) and roentgenograms were taken. The roentgenograms showed the stomach and the intestines of the fetus to be opacified. This was interpreted as showing congenital intestinal abnormality. The patient lived only one half hour after birth and at autopsy was found to have meconium peritonitis, with a giant cyst with which a portion of the terminal ileum communicated. Calcifications in the area of the fetal abdomen on the antenatal roentgenograms were not mentioned.

SUMMARY

1. The literature pertaining to the roentgen diagnosis of meconium peritonitis is reviewed briefly, with emphasis on the antenatal diagnosis.

2. A case of meconium peritonitis diagnosed prior to birth on pelvimetry films is reported. Calcium deposits were identified in the area of the fetal abdomen on lateral roentgenograms. Pelvimetry had been carried out because of marked abdominal enlargement and polyhydramnios during the early part of the last month of pregnancy. The patient was operated upon shortly after birth and recovered.

3. The case reported here is, as far as the author can ascertain, the second recorded case of meconium peritonitis in which an antenatal diagnosis was made and the first case of antenatal diagnosis of meconium peritonitis in which the infant was managed successfully by operation.

4. The lateral pelvimetry films provide the most definitive evidence of calcium deposits in the area of the fetal abdomen in cases of meconium peritonitis.

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ADDENDUM

Follow-up roentgenograms of the infant's abdomen 1 year later revealed reabsorption of the abdominal calcification. The baby has continued with normal growth and weight gain, with no further complications.

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THE HYPAQUE ENEMA AS A DIAGNOSTIC AID IN THE EVALUATION OF INTESTINAL OBSTRUCTION IN THE NEWBORN INFANT*

MECONIUM ILEUS: REPORT OF A CASE

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SUPINE and upright roentgenograms of the abdomen afford the attending physician an extremely valuable aid in the diagnosis of intestinal obstruction in the adult patient. In the newborn infant, however, the use of such roentgenograms of the abdomen to determine whether the obstruction is in the small bowel or in the colon, is frequently misleading. This is true especially if the abdominal distention is severe due to difficulty in distinguishing between small and large bowel in the infant. The use of an opaque enema (hypaque 25 per cent) is a rather simple and highly informative diagnostic procedure in such cases.

REPORT OF A CASE

B.P., a baby girl, was born on June 20, 1961, primipara with a normal full term gestation and low forceps delivery. Her weight at birth was 8 pounds and 4 ounces.

Signs and symptoms of an acute intestinal obstruction developed 24 hours after birth. Plain roentgenographic examination of the abdomen revealed a rather severe dilatation of the bowel, with intestinal contents located primarily in the lower quadrants mixed with small amounts of gas (Fig. 1). There were no abnormal densities to suggest the presence of peritoneal calcifications. The rectum and sigmoid were devoid of air, but some of the severely distended loops of bowel in the upper quadrants were thought to be possibly colon. Rectal examination revealed an empty rectum.

Because the possibility of a colon stenosis or atresia, a hypaque (25 per cent) enema was given to the infant under fluoroscopic control by means of a small rectal catheter and a Triumph syringe. The opaque enema study revealed a collapsed and ribbon-like colon (Fig. 2). A preoperative diagnosis of a meconium ileus was established.

Operative Findings. On surgical exploration, the ileum was found to be extremely distended proximal to a loop and containing a rather thick meconium in the terminal ileum. The colon was empty and collapsed and there was no evidence of perforation. There was a small amount of free peritoneal fluid. About 12.5 cm. of the terminal ileum was resected and a Mickulicz's double barrel ileostomy was performed. The infant tolerated the operation well.

Surgical Pathology. On section and microscopic study of the surgical specimen, the lumen was found to be filled with an eosinophilic amorphous material in which keratotic cells, probably vernix, were present. The mucosa and the walls appeared normal. The serosa had focal areas of edema, congestion, and mild inflammatory infiltrates. The anatomic diagnosis was meconium ileus.

Postoperative Course. The immediate postoperative recovery was uneventful. On July 4, 1961, however, signs and symptoms of intestinal obstruction recurred, although the ileostomy appeared to be functioning well. Persistent inability of the infant to tolerate oral feedings was evaluated by means of a hypaque swallow, but no evidence of gastric retention or intestinal obstruction could be demonstrated.

The infant continued to lose weight despite intravenous feedings, blood transfusions, and repeated irrigations of the ileostomy with enzymatic solutions (viokase and caroid) in saline. Although the electrolyte balance was well maintained, hydration could not be stabilized. On July 26, 1961, the infant appeared critically ill, and plain roentgenograms again revealed evidence of intestinal obstruction. The chest was clear to auscultation and roentgenographically. The infant expired on August 4, 1961.

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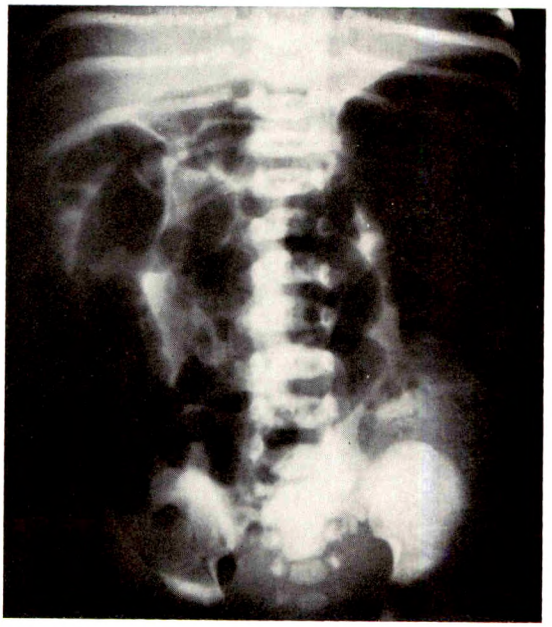


FIG. 1. Greatly distended loop of bowel in the left upper quadrant thought to be colon. Note meconium mixed with gas in the left lower quadrant.

Necropsy Findings. On postmortem examination, a gangrenous loop of ileum was found, as a result of postoperative adhesions, with peritonitis and terminal bronchopneumonia. The gross appearance of the pancreas was normal. On microscopic study of sections of the pancreas, however, most of the pancreatic ducts appeared dilated. Almost all of the ducts contained a laminated eosinophilic material which resembled fibrin and was acellular. The epithelial lining was flattened. The number of acini was markedly reduced and the interstitial connective tissue was increased (Fig. 3). The liver revealed on section and microscopic study many small bile canaliculi containing bile thrombi, more numerous in the portal areas. Microscopic study of sections of the lungs revealed the bronchi filled with neutrophils, fibrin, and desquamated epithelial cells. The walls of the bronchi were infiltrated with round cells, but these did not penetrate into the alveoli (Fig. 4).

The anatomic diagnosis was cystic fibrosis of the pancreas.

DISCUSSION

Intestinal obstruction in the newborn infant, due to meconium ileus, is extremely rare and generally a surgical emergency with a rather poor prognosis and high operative mortality.[2,11,15] Meconium ileus accounts for 7 to 15 per cent of all cases of intestinal obstruction in the newborn infant.[11] At Touro Infirmary in the last 12 years, out of 242,183 admissions and 27,568 deliveries, there have been 2 verified cases of meconium ileus, both ultimately fatal, including the one reported here.

The etiology and pathology of meconium ileus have been extensively studied and it is generally agreed that pancreatic achylia is probably the main precipitating factor.[5,8,15] Occasionally, a case of so-called meconium ileus without evidence of pancreatic fibrocystic disease has been reported.[6] The highly viscid nature of the meconium found in these cases of intestinal obstruction is due to its large content of albumin and globulin. The electrophoretic patterns of the abnormal meconium and of the amniotic fluid as studied by Young *et al.*[17] revealed identical characteristics. The absence of the exocrine secretions of the pancreas makes impossible the hydrolization of the above mentioned proteins from the ingested amniotic fluid, which together with the normal components of meconium are concentrated by water ab-

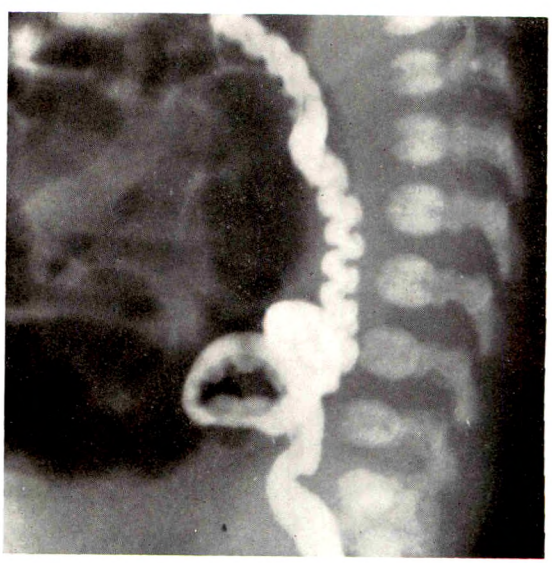


FIG. 2. Opaque enema study shows a collapsed, ribbon-like colon.

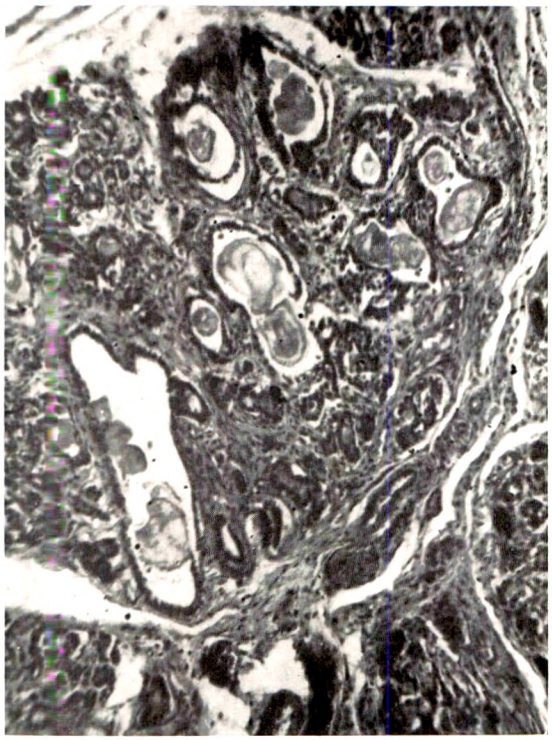


Fig. 3. Photomicrograph of the pancreas showing that the number of acini is markedly reduced and the interstitial connective tissue is increased. The dilated ducts contain laminated eosinophilic material.

sorption to produce the thick and sticky meconium.

Plain roentgenographic examination of the abdomen of the newborn infant with intestinal obstruction is sometimes not sufficient to establish the diagnosis of small bowel obstruction and the opaque enema study is suggested by many authors in order to exclude other conditions which are not necessarily surgical emergencies and in which surgery may not be the treatment of choice.[3,4,7,9,12–16] The only fairly characteristic and frequent roentgenographic finding in cases of intestinal obstruction in the newborn infant due to meconium peritonitis is the presence of peritoneal calcifications.[1,10] Other conditions leading to intestinal obstruction in the newborn which are not surgical emergencies or in which definitive surgical treatment is best deferred and which have symptoms and signs similar to meconium ileus are the meconium plug syndrome,[4] colo-colic or ileo-colic intussusception,[9,11,14] and Hirschsprung's disease.[3,16] These conditions can be readily diagnosed with the simple opaque enema study, and at times an unnecessary surgical exploration may be avoided. We prefer the hypaque solution because this is a water soluble opaque medium which will not interfere with immediate surgical exploration and will not cause fecal impactions.

In the cases of a meconium plug syndrome, the opaque enema examination will locate the plug, which is frequently expelled with the enema, and which if not expelled can be repeatedly washed with hydrogen peroxide solutions and facilitate expulsion.

In the cases of congenital megacolon, the aganglionic segment may be located by the opaque enema examination; and if in the rectum, a simple rectal intubation will relieve the obstruction temporarily, until more definitive surgical treatment can be performed. In the cases of intussusception, the opaque medium also will locate the

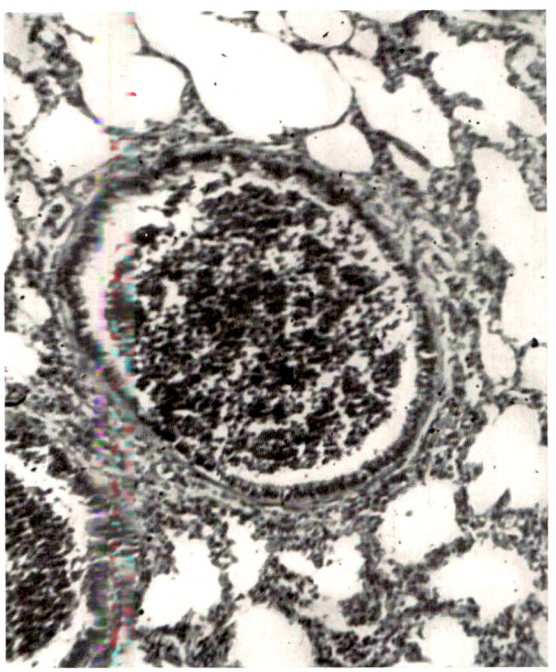


Fig. 4. Photomicrograph of the lung showing the bronchi filled with neutrophils, fibrin, and desquamated epithelial cells.

intussusceptum, and at times relieve the obstruction without surgical exploration.

SUMMARY

A case of meconium ileus is presented. The use of the hypaque enema study is suggested in all cases of intestinal obstruction in the newborn infant to avoid the possibility of unnecessary surgical exploration in cases which may be cured or relieved by the enema or in which surgical treatment may be well deferred until the infant can better tolerate surgery.

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Vol. 38, No. 3

TOXIC DEPRESSION OF THE NEWBORN CAUSING DEFICIENT INTESTINAL GAS PATTERN*

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PAUCITY or absence of intestinal gas in the newborn period, commonly attributed to mechanical obstruction, have also been described in nonobstructive disorders.[1,3,4] Feinberg, Margulis, and Nice[3] outlined the following causes of deficient intestinal gas patterns: (1) mechanical obstruction; (2) mechanical obstruction associated with dehydration; (3) dehydration from causes unrelated to obstruction; and (4) interference with normal gas transit due to respiratory distress.

We have recently observed 3 newborn infants with deficient intestinal gas patterns who are of particular interest. These infants were victims of the depressant effects of drugs administered to their mothers during labor. Awareness of the nonobstructive disorders associated with deficient intestinal gas patterns in the newborn resulted in an accurate roentgenologic diagnosis.

REPORT OF CASES

CASE I. A full term infant weighing 3,340 gm. was delivered spontaneously 8 minutes after the mother received an intravenous injection of 50 mg. of demerol and 25 mg. of phenergan. After delivery the child cried spontaneously and the tracheobronchial passages were aspirated by nasopharyngeal suction.

On admission to the nursery, the infant was limp, had a weak, high pitched cry, and regular, shallow respirations. A bluish discoloration of the skin was noted. The pupils were constricted. A diagnosis of idiosyncrasy to opiates was made and the infant was treated with nalline and coramine. A short period of improvement was followed by deep narcosis, Cheyne-Stokes breathing and failure to respond to painful stimuli. Oxygen and respiratory stimulants were continued.

Twenty-four hours after birth, a roentgenogram of the chest and abdomen was made (Fig. 1*A*). A small amount of gas was seen within the stomach. No gas was demonstrated distally. Several hours later, a second roentgenogram demonstrated a small amount of gas in the stomach and in several loops of the small intestine (Fig. 1*B*). Soft tissue densities in both lower lung fields were visualized on both roentgenograms. Diagnoses of neonatal pneumonia and deficient intestinal gas pattern resulting from respiratory depression were made.

The infant improved slowly, but respirations and active feeding remained poor for several weeks. Two weeks after birth, a third roentgenogram (Fig. 1*C*) showed a moderate amount of gas in the stomach and colon and practically no gas within the small intestine. The intestinal gas pattern was described as diminished. The lung fields were clear.

The infant continued to improve and was discharged 5½ weeks after birth. A follow-up roentgenogram was not obtained. The discharge diagnosis was idiosyncrasy to opiates and deficient intestinal gas pattern due to respiratory failure.

CASE II. A premature infant of 35 weeks' gestation was delivered by cesarean section. Four hours prior to delivery the mother had been sedated for eclampsia with 6 gr. of nembutal intravenously. At birth, the infant's breathing was slow and shallow and he lacked an adequate swallowing reflex.

Thirty-six hours after birth, a roentgenogram of the abdomen revealed a small amount of gas in the intestinal loops in the right mid-quadrant (Fig. 2, *A* and *B*). This was interpreted as a deficient intestinal gas pattern related to poor respiration.

On the following day, the child died. Autopsy revealed bronchopneumonia. The intestinal tract was normal.

CASE III. A premature girl of 35 weeks' gestation, weighing 2,100 gm., was delivered spon-

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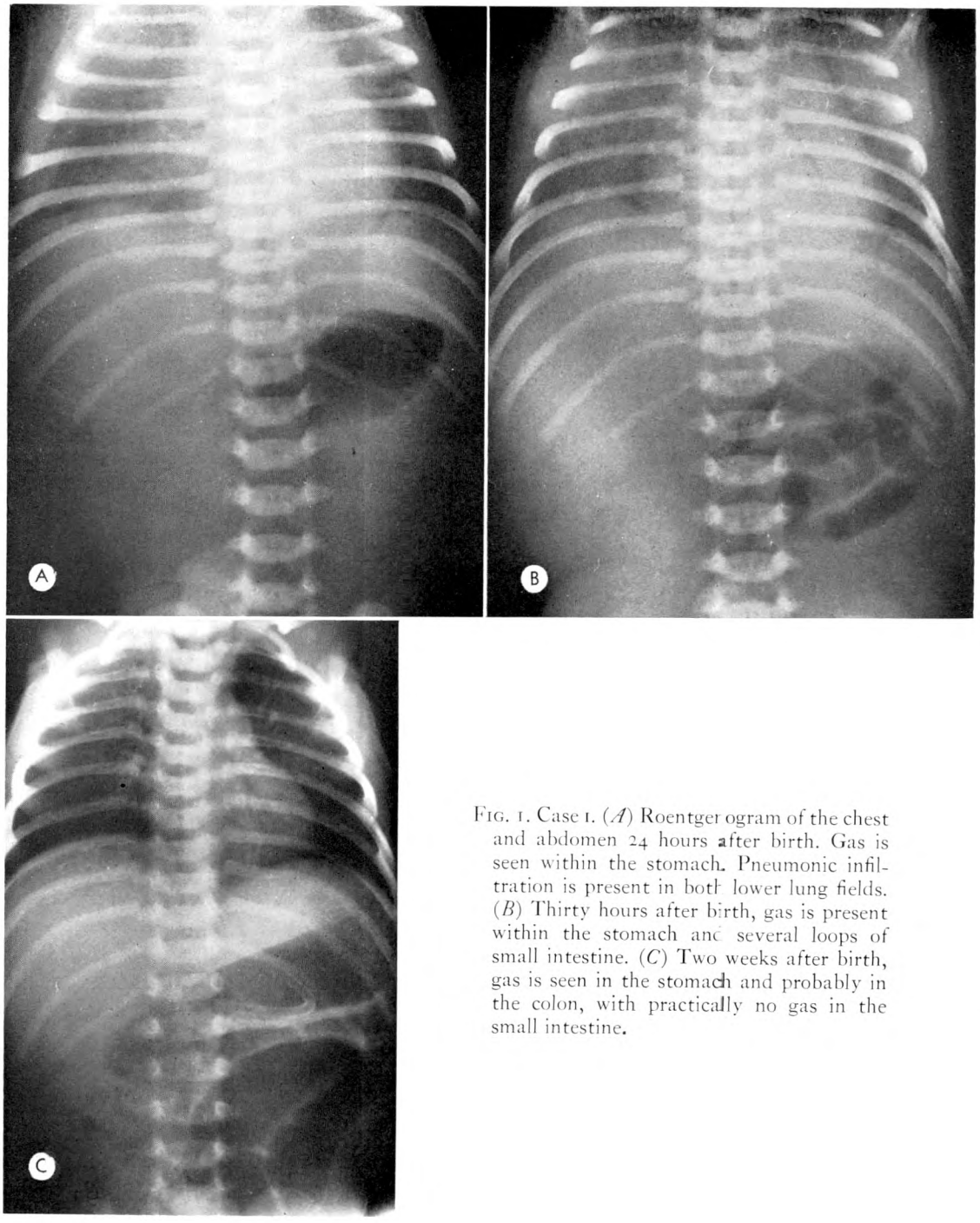


FIG. 1. Case 1. (*A*) Roentgenogram of the chest and abdomen 24 hours after birth. Gas is seen within the stomach. Pneumonic infiltration is present in both lower lung fields. (*B*) Thirty hours after birth, gas is present within the stomach and several loops of small intestine. (*C*) Two weeks after birth, gas is seen in the stomach and probably in the colon, with practically no gas in the small intestine.

taneously. The mother had been given 50 mg. of demerol and 25 mg. of phenergan intravenously 2 hours prior to delivery. The medication had been repeated (in half dosage), ½ hour prior to delivery. The infant was apneic for 5 minutes after birth. Positive pressure mouth-to-mouth breathing was performed. The child responded and was transferred to the nursery.

In the nursery, the infant was noted to have rapid, shallow respirations with intercostal re-

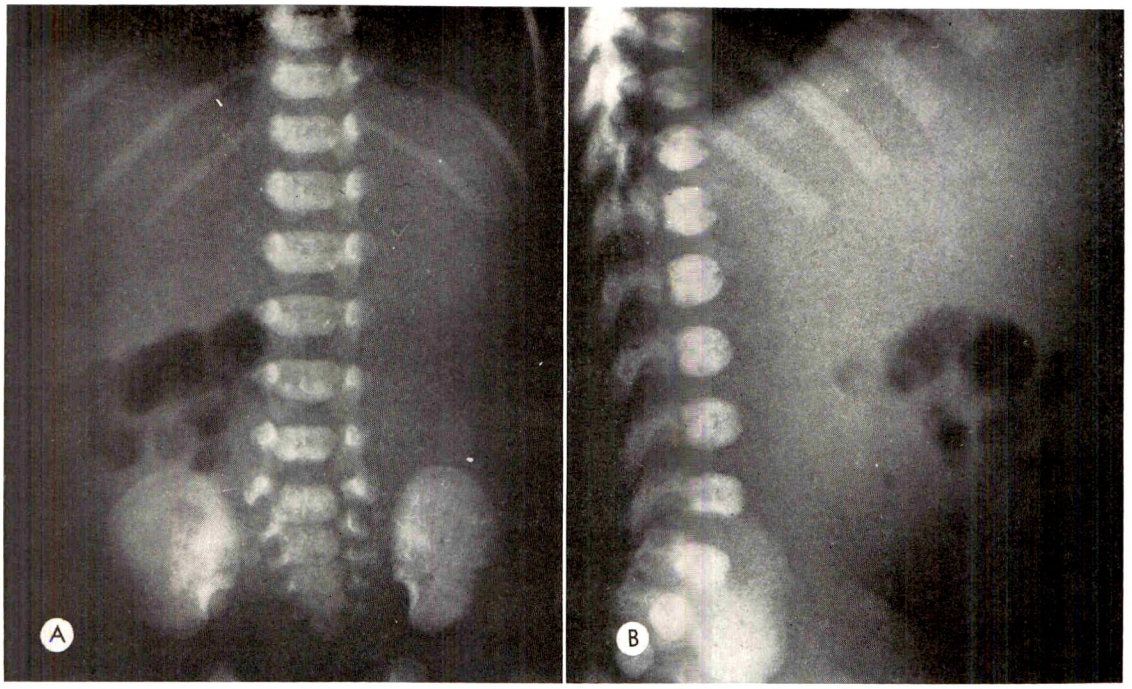


Fig. 2. Case II. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the abdomen demonstrate a small amount of intestinal gas in the right mid-quadrant, 36 hours after birth.

tractions. Feeble breath sounds were audible in the upper lung fields; none were heard in the bases.

A roentgenogram 12 hours after delivery (Fig. 3) showed a small amount of gas in the stomach and probably in the colon. Little gas was identified in the small intestine. A deficient intestinal gas pattern due to poor respirations was reported.

Examination of the infant's blood revealed a hemoglobin concentration of 9.5 gm./100 cc. Increased fetal hemoglobin in the maternal blood indicated that fetal bleeding into the mother had occurred. The infant was transfused with 200 cc. of packed red blood cells. She subsequently improved and was discharged.

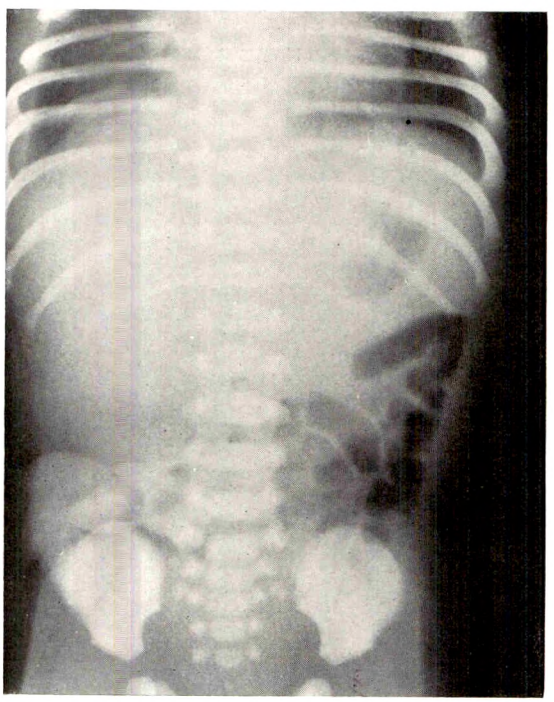


Fig. 3. Case III. Roentgenogram of the abdomen 12 hours after delivery reveals a small amount of gas in the stomach and probably in the colon. Little gas is seen in the small intestine.

DISCUSSION

With the first breath of the newborn, air is believed to enter the esophagus and stomach by the creation of negative pressure.[2] Air may enter the esophagus and stomach, even before the first breath, by active sucking movements or swallowing.[3,4] Roentgenograms of the abdomen, taken immediately after birth, reveal gas to be

present normally in the stomach.[3,4,6,7] Progression of intestinal gas results in complete filling of the small intestine in 2 hours and of the large and small intestine, in 3 to 6 hours.[3,4,6,7] Crying was found to have no effect on the rate of progression of intestinal gas.[4]

Feeble and premature infants, as well as infants in severe respiratory distress, are found in many instances to have delayed appearance of normal intestinal gas or diminished intestinal gas.[3,4] The disturbance of normal swallowing, accompanying respiratory distress, is thought to be responsible for the abnormal gas pattern.[3] The newborn infants herein reported had respiratory depression from medication given to the mother during labor.

The effects of barbiturates and opiates on the newborn have been reviewed by James.[5] Barbiturates and opiates have a twofold effect on the fetus *in utero*. The maternal-fetal oxygen-carbon dioxide exchange is altered when the maternal respiratory center is depressed. In addition, these drugs pass freely through the placenta to depress the fetus. Normally, the intrauterine oxygen levels of the fetus are believed to be high, but they may be compromised by drug depression *in utero*. In the absence of drugs, there is a marked reduction in oxygen-carbon dioxide exchange between mother and newborn during labor and delivery. This biochemical birth asphyxia may vary considerably with the degree of birth trauma. Therefore, the clinical condition of the newborn depends on the depressant effect of drugs *in utero*, physiologic asphyxia of the birth process, and the physical trauma of birth. The greatest potential danger lies in biochemical birth asphyxia superimposed on a high level of depressant drugs. In this situation, the action of administered opiates and barbiturates may progress from depression to toxicity.

Awareness of the possibility of toxic depression of the newborn resulting in diminished or deficient intestinal gas may prevent needless operations for presumed mechanical obstruction.

SUMMARY

A deficient intestinal gas pattern on a nonobstructive basis is described in 3 newborn infants. Respiratory depression, caused by administration of drugs to the mother during labor, is believed to be responsible for the abnormal gas pattern. Recognition is necessary for proper management of the newborn.

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Vol. 88, No. 3

DISTAL URETERAL ATRESIA*

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CAMPBELL states that distal ureteral atresia with cyst formation is rarely diagnosed prior to operation.[1] Several reported cases seem to support this contention.[5,6,8] However, certain clinical and roentgenologic features, if recognized, should make correct preoperative diagnosis more frequent.

Various terms have been used to describe this condition. Campbell[1] alternately labels it ureteral agenesis, ureteral hypoplasia, or blind ending ureter. Slater[8] called it ureteral atresia, thereby implying a more specific malformation consisting of imperforation or absence of a normal opening. Regardless of its name, the condition simply represents failure of the distal ureter to communicate with the bladder or any other structure. Instead, it terminates as a cul-de-sac adjacent to the bladder. The remaining proximal ureter may become the site of a large cyst. Thus, the clinical problem becomes one of differential diagnosis of abdominal tumors. The inverse relationship between the frequency of correct preoperative diagnosis and the incidence of operative morbidity and mortality clearly demonstrates the need for thorough investigation and accurate diagnosis prior to operation.[4]

EMBRYOLOGY

Approximately 10 per cent of all individuals are born with urogenital malformations which comprise about 35 to 40 per cent of all congenital anomalies.[1] The ureter is the commonest site of upper urinary tract malformation, and all lesions there are potentially obstructive. The most frequent ureteral anomaly is unilateral duplication, either complete or incomplete. Campbell[1] found 301 patients with complete ureteral duplication in 47,409 autopsies. About one-third had an additional urologic malformation, and in 19, it occurred on the side opposite the ureteral duplication. Since 1956, 3 patients with distal ureteral atresia were operated upon at Children's Hospital of Michigan; all 3 had unilateral duplication of the kidney and ureter. In 2, atresia and duplication were on the same side, while they occurred on opposite sides in the other.

The pathogenesis of distal ureteral atresia is not known. However, there are two aspects of fetal ureteral development[3,9] which provide bases for speculation.

Normally ureteral buds arise from the wolffian duct during the fifth or sixth week of fetal life. They originate from the posterior aspect of the duct in the region where it turns sharply to enter the cloaca. The buds then grow cephalad and fuse with the renal blastema. During the seventh week, the wolffian ducts and ureters become incorporated into the inferior portion of the bladder anlage.

Complete duplication of the ureter results when two ureteral buds arise simultaneously. The exact level at which the primary or accessory bud arises is probably variable. Hawthorne[3] has postulated that if the cephalad bud arises too high on the wolffian duct, it may fail to be incorporated into the trigone area during the phase of development when the wolffian duct is drawn into the bladder to form its base. Presumably the same thing would apply if only a single bud were involved. The type of ureteral abnormality that results depends upon the subsequent fate of the wolffian duct remnant. In the female the duct ordinarily undergoes degeneration, thus a ureter which terminates blindly would result. But in the male where the wolffian duct remnant is the precursor of

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other urogenital structures, Hawthorne's hypothesis would explain such ectopic ureteral orifices as those in seminal vesicles, posterior urethra, vas deferens, or epididymis; but it would not explain a ureter with distal atresia.

Thus one must search for another explanation for its occurrence in both sexes. Chwalla,[2] in 1927 reported that in the 10 to 12 mm. stage of embryonic development, the ureteral bud, as it originates from the wolffian duct, becomes closed over by an epithelial membrane consisting of two layers of cells. This membrane is present in all fetal ureters and usually persists until the 18 to 24 mm. stage. It is usually absorbed then but occasionally it persists longer and, in one instance Chwalla observed its presence in a 104 mm. embryo. As the time of disappearance may vary, it is plausible to surmise that occasionally it may not disappear at all, resulting in an imperforate or atretic distal ureter regardless of fetal sex. This theory is supported by Vermooten[9] who attributes most congenital obstructive conditions in the distal ureter to persistence of this membrane in part, or in whole.

Either theory explains the occurrence of atresia in a female, but abnormal persistence of a fetal membrane seems to be the only explanation for atresia in males.

REPORT OF CASES

CASE I. This 10 month old infant was noted to have an enlarged abdomen since birth. She became acutely ill 2 days prior to hospitalization with persistent vomiting. There was a past history of recurrent unexplained febrile episodes. On admission she was noted to have an extremely large abdomen. Her temperature was 100.8°F. A large smooth mass was palpable in the left upper quadrant and extending down into the pelvis. It was of cystic character with a detectable fluid wave. Rectal examination revealed the mass lying anteriorly and to the left.

Laboratory studies showed a normal urinalysis; hemoglobin 8.1 gm. per 100 ml.; blood urea nitrogen 12 mg. per 100 ml.

Pertinent roentgenographic findings were a retroperitoneal, lobulated mass extending from the pelvis into the left upper quadrant. An intravenous pyelogram showed no function on the left; the right side showed complete ureteral and partial renal duplication but was otherwise normal; and the bladder was displaced anteriorly and laterally into the right lower quadrant. The rectovesical space was greatly widened.

Operation revealed a small left kidney with a grossly dilated cystic ureter terminating blindly at the level of the bladder floor. It contained a large quantity of clear fluid. The bladder was opened but no left ureteral orifice was identified. A left ureteronephrectomy was done but for technical reasons a few inches of the distal ureter was not removed. A Foley catheter was placed in this and the residual ureteral pouch was sutured about the catheter. Several days later opaque medium was instilled into the remaining ureter through the catheter and roentgenograms were made. These demonstrated no communication with the bladder or any other pelvic structure. Following drainage of the medium from the ureteral pouch, a cystogram was made. This revealed no intrinsic bladder abnormality and no evident reflux into the remaining ureteral segment.

The pathologist reported hypoplasia of the left kidney. The collapsed ureter measured 30 cm. × 3 cm. and was lined by foci of atrophic as well as hyperplastic epithelium. The muscular coat was hypertrophied and chronic inflammatory cells were present in the lamina propria.

CASE II. This 4 month old infant was hospitalized because of a possible rotary nystagmus. On admission his temperature was 101°F., his abdomen appeared distended and there was a cystic, smooth left flank mass extending to the midline and from the left costal margin down into the pelvis.

Laboratory studies showed a normal urinalysis; hemoglobin 9.7 gm. per 100 ml.; blood urea nitrogen 23.9 mg. per 100 ml. on admission and 16.9 mg. per 100 ml. one week later.

Pertinent roentgenographic findings were a lobulated, sharply defined, retroperitoneal mass extending from the pelvis into the left flank area. An intravenous pyelogram showed a normal right upper urinary tract; the visualized left collecting system showed pyelocaliectasis and was displaced laterally, upward and anteriorly; the left ureter was displaced anteriorly; and there was smooth extrinsic pressure deformity of the bladder which was displaced

anteriorly and laterally into the right lower quadrant. The rectovesical space was increased in width (Fig. 1, *A* and *B*). Extensive enlargement of the entire length of a completely duplicated left ureter was postulated as the most probable anatomic explanation for the observed deformities.

Operation revealed a cystic appearing lower pole of the left kidney with a large cystic ureter extending from it down to the bladder area but without evident communication between the bladder and dilated ureter. A heminephrectomy and ureterectomy were performed.

The pathologist reported that the presumed lower pole cyst was probably a dilated renal pelvis measuring 2 cm. in greatest diameter. The collapsed ureter measured 16 cm. by 1.5 cm.

Case III. This 4 month old infant was well until one week prior to hospitalization when she developed persistent fever and colicky abdominal pain. On admission her temperature was 100.8°F.; her abdomen appeared distended and there was a tender, large, fluctuating mass which could be palpated in her left flank. It extended down into the pelvis where, on rectal examination, it could be felt to lie anteriorly and to the left.

Laboratory studies showed a normal urinalysis; hemoglobin 8 gm. per 100 ml.; blood urea nitrogen 7.5 mg. per 100 ml.

The significant roentgenographic findings were a retroperitoneal mass extending from the pelvis into the left flank. An intravenous pyelogram showed a normal right kidney and ureter; the visualized left renal collecting system was normal in appearance except for displacement laterally, downward, and anteriorly; the left ureter was displaced anteriorly; and a smoothly marginated extrinsic pressure deformity of the bladder dome was evident with anterior displacement. Considerable increase in the rectovesical space was present (Fig. 2, *A* and *B*). Distal atresia of a completely duplicated ureter with massive dilatation of the entire proximal portion was considered the most likely basis for the visualized abnormalities.

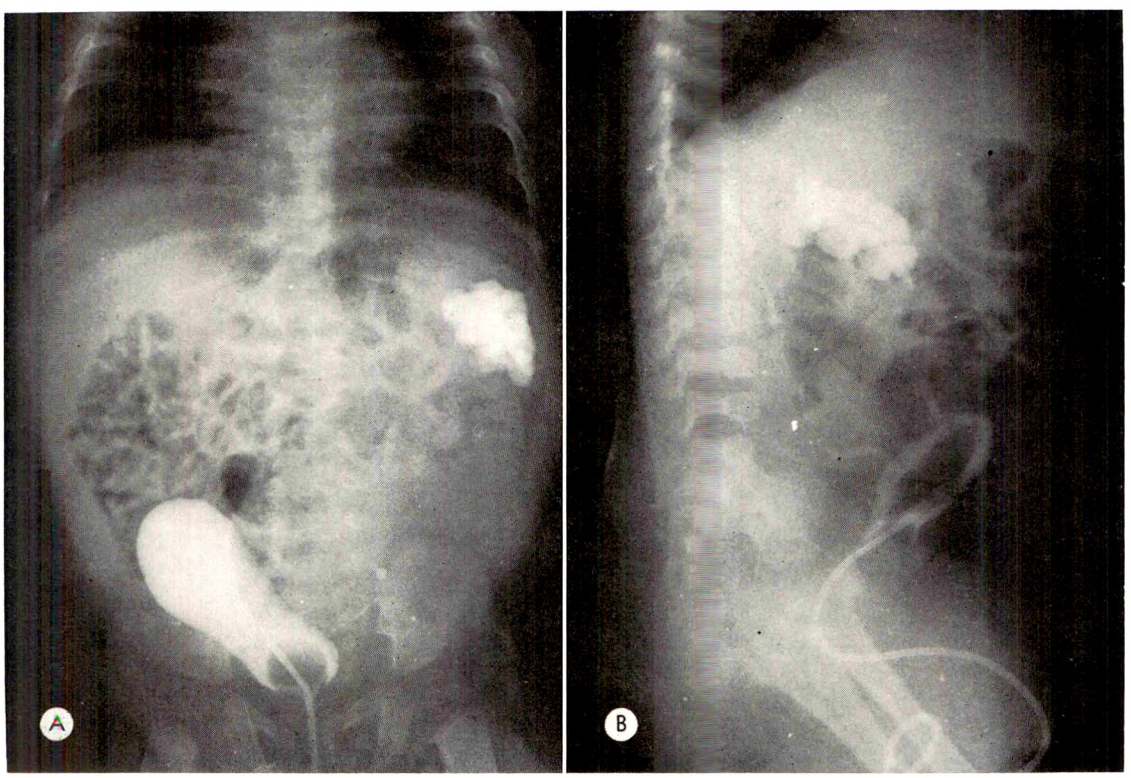


Fig. 1. (*A*) Intravenous pyelogram shows left pyelocaliectasis with renal, ureteral and bladder displacement and distortion by an extensive mass. (*B*) Lateral pyelogram shows the mass to be retroperitoneal and demonstrates increase in the space between the bladder and the rectum.

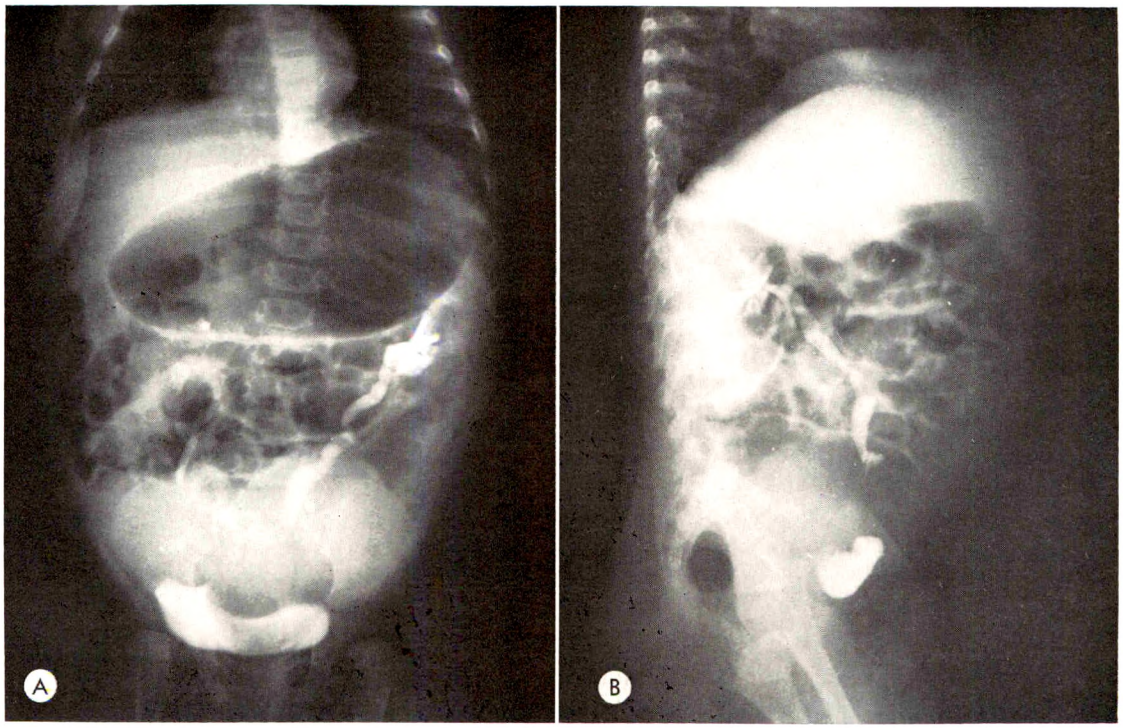


FIG. 2. (*A*) Intravenous pyelogram shows displacement of the left kidney and ureter as well as an extrinsic pressure deformity of the bladder by a large mass. (*B*) Lateral pyelogram shows that the mass is retroperitoneal and, besides displacing the kidney and ureter, it produces an increase in the space between the rectum and the bladder.

Operation revealed a dilated, cystic ureter arising from an atrophic upper pole of the left kidney and extending down to the bladder; it showed no evidence of ureterovesical communication. A heminephrectomy and ureterectomy were performed. The ureter contained viscid pus which, on culture, showed *Escherichia coli* organisms.

The pathologist reported evidence of acute and chronic inflammatory disease in the atrophic renal tissue. The collapsed ureter measured 26 cm.×3 cm. Its smooth muscle coat was hypertrophied and inflammatory infiltrates were present within its walls.

DISCUSSION

CLINICAL MANIFESTATIONS

Infants and children may or may not have signs or symptoms of sufficient magnitude to arouse clinical concern; thus the condition may go unrecognized until adulthood, or conceivably remain undetected throughout life.

When distal atresia is discovered in early childhood the presenting symptoms are invariably related to cystic dilatation of the ureter proximal to the atresia. The cyst can be so large that it simulates an extensive abdominal tumor. History usually reveals that the child has had a large, protuberant abdomen since birth. No other signs or symptoms need be present although often the child has had low grade, febrile episodes without any evident focus of infection which subsided with antibiotic therapy. Presumably, these episodes precede the development of a pyo-ureteronephrosis secondary to infection in the renal pelvis drained by the cystic ureter. When this occurs, the fever persists despite treatment and crampy abdominal pain may coexist.

Physical examination reveals a cystic mass which extends from the pelvis into the upper abdomen or flank area. It can also be palpated by rectal examination and

found anteriorly; it may be tender if acute inflammation is present.

Laboratory findings are of little positive value; however, one negative result is of major significance: absence of pyuria. When true atresia is present the urine reaching the bladder will be free of pus or bacteria.

The condition is much less apparent in adults because although the cystic ureter enlarges with growth, the abdominal cavity shows greater relative increase in size; proportion obscures the distention. Thus identification of this entity in an adult usually results either from accidental discovery of a mass or from investigation of persistent fever and subsequent discovery of an abdominal or pelvic mass.

ROENTGENOGRAPHIC FINDINGS

The clinical and laboratory findings may result in an increased index of suspicion, but roentgen examination contributes most to correct preoperative diagnosis.

Plain roentgenograms of the abdomen reveal a large, usually sharply defined mass extending from the pelvis into one of the upper abdominal quadrants; if well visualized the mass may appear lobulated. The renal and psoas muscle shadows are indistinct on the involved side. The bowel loops are displaced upward from the pelvis.

The mass exhibits additional features of space occupying retroperitoneal lesions:[7] the descending or ascending colon is displaced anteriorly and medially as are the small bowel loops; the sigmoid colon appears straightened and displaced anteriorly.

Of greatest value is the excretory pyelogram. The appearance here varies, depending on the presence or absence of associated duplication on the involved side. In our patients the kidney connected with the atretic ureter showed no evidence of function; thus the ureteral mass did not opacify. If, as in 2 of our patients, there is associated homolateral duplication, the unaffected ureter and its renal tissue will usually visualize. The duplicated collecting system may show mild dilatation or have a normal appearance except for displacement of the renal tissues by the enlarged ureter. Displacement is usually lateral, but its upward or downward direction depends on whether the atretic ureter drains the inferior or superior pole of the kidney. The patent duplicated ureter follows a tortuous course because the cystic ureter displaces it at various levels. The bladder shows extrinsic pressure deformity, is displaced away from the mass, and often demonstrates elevation of its base. Such bladder changes are present regardless of whether or not a homolateral duplication exists.

The most characteristic findings are shown on a lateral excretory pyelogram. If duplication exists, the functioning homolateral collecting system and ureter will be displaced anteriorly on the pyelogram. The outstanding feature, however, is the great increase in the space between the bladder and the rectum produced by the mass. The bladder shows posterior extrinsic pressure with resultant anterior displacement; similar pressure deformity on the anterior rectal wall is also present, but is evident only when there is enough gas to adequately distend its lumen. The increase in space between the bladder and rectum cannot be demonstrated in frontal projection.

When considered separately, each of these roentgenographic findings is nonspecific, but distortion of so great an anatomic area by one mass is highly unusual and not characteristic of any other lesion. Since the pyelogram shows the mass to be intimately related to the entire upper urinary tract, one should suspect extreme ureteral enlargement, as no other condition produces such extensive bladder and renal alterations simultaneously.

DIFFERENTIAL DIAGNOSIS

Ureteral enlargement to this extent is unique to distal ureteral atresia. It does not exist with valves, strictures or ureteroceles, and in addition the ureter associated with these defects is usually visualized on ex-

cretory pyelograms. Moreover, these partially obstructing lesions are invariably associated with chronic pyuria; this is never found with atresia of the distal ureter.

Other retroperitoneal tumors such as renal cysts, hydronephrosis, renal embryoma, teratoma, and neuroblastoma may produce similar abdominal or pelvic findings separately, but not simultaneously. In addition, each of these has certain characteristics of its own which serve to differentiate them further.

Ovarian cysts and hydrometrocolpos could conceivably produce similar bladder and rectal deformities but they do not cause the abdominal changes associated with retroperitoneal tumors.

CONCLUSIONS

The major clinical problem associated with atresia of the distal ureter arises when the ureter proximal to the atretic end becomes the site of cystic dilatation, and this mass must be differentiated from other abdominal tumors. The distinguishing clinical features are: (1) history of a protuberant abdomen dating from infancy, (2) recurrent unexplained febrile episodes, (3) presence of an extensive cystic mass which occasionally is tender, and (4) absence of pyuria. The most significant roentgenographic finding is a retroperitoneal mass involving the area from the pelvis to the upper abdomen, closely associated with the upper urinary tract, and producing simultaneous changes in the bladder, rectum, kidney, and ureter.

Using these criteria, an accurate preoperative diagnosis of distal ureteral atresia should not prove to be the rarity it has been in the past.

SUMMARY

Three case histories of 3 patients with distal ureteral atresia and secondary cystic dilatation of the proximal ureter are presented. The distinguishing clinical and roentgenographic features are discussed as well as the probable embryologic errors in development producing this condition.

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Vol. 88, No. 3

CONGENITAL DISSEMINATED LUPUS ERYTHEMATOSUS*

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COLLAGEN diseases such as disseminated lupus erythematosus, polyarteritis, systemic scleroderma and dermatomyositis are relatively infrequent in childhood. All collagen diseases, including rheumatic fever, are rare before the age of 3 years. There are several reports which draw attention to a familial connection[1,2] in patients with disseminated lupus erythematosus, including 2 reports in identical twins.[11]

The lupus erythematosus (L.E.) factor is transmitted through the placenta.[3,4] The cord blood of newborn infants of mothers with disseminated lupus erythematosus will sometimes contain a high level of gamma globulin and may contain L.E. cells. Usually the infant is asymptomatic and the L.E. phenomena disappear within a few months. In one reported case (confirmed at autopsy) the infant apparently was born with a clinical picture compatible with disseminated L.E. and endocardial fibroelastosis.[7]

We have observed a unique case at Charity Hospital. The infant was born with signs of disseminated lupus erythematosus, although there was no history of any collagen disease in either parent or in 2 siblings.

REPORT OF CASE

This Negro female infant was born on March 19, 1957, the third child of a 29 year old mother who had no complications during pregnancy. The birth weight was approximately 3,700 gm. (7 lb., 12 oz.).

At birth the infant had numerous petechiae on the forearms, mild conjunctivitis and a subconjunctival hemorrhage in the medial portion of the right eye. A low platelet count, positive Coombs' test and a positive L.E. cell preparation led to a diagnosis of congenital disseminated lupus erythematosus soon after birth. The patient was followed by the Tulane Hematology Clinic. She had severe anemia with high output cardiac failure and required numerous transfusions, sometimes as often as every 1 or 2 weeks for considerable periods of time. The patient developed bronchopneumonia on several occasions and on numerous clinic visits purpuric eruptions were noted. On one occasion a diagnosis of measles was made.

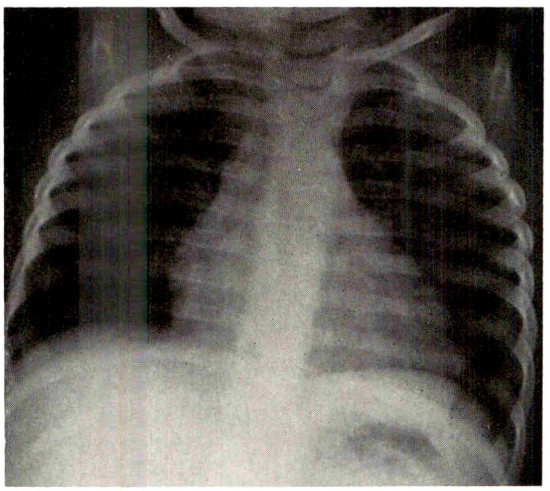


FIG. 1. April 27, 1958: Heart and lungs are relatively normal. Minimal cardiac enlargement may be present.

During the first year of life roentgenograms of the heart and lungs were relatively normal (Fig. 1). Meticorten, 15 mg. per day, was given from April to October, 1959. In August, 1959, it was noted that the spleen was becoming larger (Fig. 2) and in September, 1959, cardiac enlargement with pulmonary shadows due to edema or pneumonia was demonstrated (Fig. 3).

In November, 1959, a splenectomy was done and the meticorten dosage was increased up to 60 mg. per day, then gradually decreased to 10 mg. per day. Postoperative atelectasis of the right upper lobe resolved within a few days. The

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Presented at the Fourth Annual Meeting of the Society for Pediatric Radiology, Miami Beach, Florida, Sept., 25, 1961.



Fig. 2. August 6, 1959: An enlarged spleen and moderate ileus are demonstrated.

need for transfusions decreased following splenectomy but was still required every 3 to 8 weeks. Intermittent steroid therapy was employed.

Beginning on April 17, 1960, numerous electrocardiograms showed T-wave changes and on March 17, 1961, there were signs of myocardial disease. A chest roentgenogram about this time revealed cardiac enlargement and bilateral pulmonary shadows, probably due to edema (Fig. 4). On September 19, 1960, there were palpable inguinal lymph nodes, but at no other time was this noted in the patient's chart.

The fifty-second hospital admission of this child was on May 20, 1961. At this time the hemoglobin was 3.5 gm., white blood cell count 12,900 with 61 per cent polymorphonuclears, and 34 nucleated red blood cells were counted per each 100 white blood cells. The urine had a specific gravity of 1.011 but was otherwise negative. The heart was dilated and there was bilateral pulmonary edema.

The patient expired on May 21, 1961. The final diagnosis was (1) disseminated lupus erythematosus, congenital; (2) hemolytic anemia, probably secondary to lupus erythematosus; and (3) congestive heart failure, secondary to anemia. Autopsy was not permitted.

DISCUSSION

Although the etiology of lupus erythematosus remains unsettled, it appears likely that it should be included among the auto-immune disorders.[5] The hematologic abnormalities include the L.E. factor; positive Coombs' test, with or without hemolytic anemia; leukopenia, at times severe; a positive serologic test for syphilis, and a hemorrhagic disorder due to circulating anticoagulants.

There is a 30 per cent fetal mortality in mothers with acute disseminated lupus erythematosus.[6] The infants usually do not have the clinical and pathologic changes of disseminated lupus erythematosus, but the hematologic phenomena such as elevated gamma globulin and the L.E. factor may be transmitted through the placenta.

Larsson and Leonhardt[8–10] reported a family of 14 siblings in which 3 had disseminated lupus erythematosus with high gamma globulin values and one member

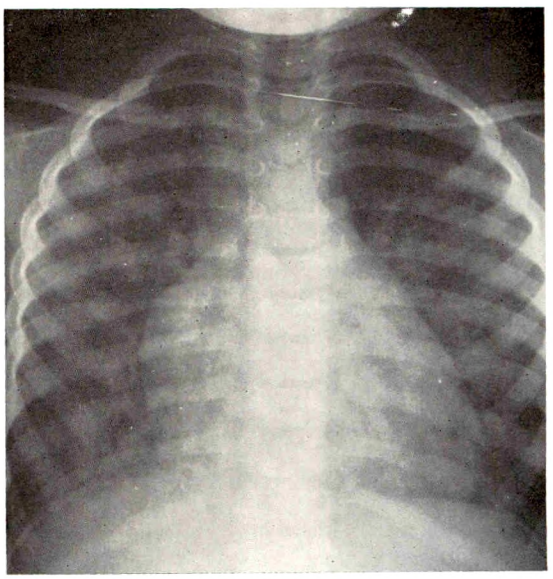


Fig. 3. September 20, 1959: Chest roentgenogram shows moderate cardiac enlargement and interstitial changes, possibly pneumonia. Some degree of pulmonary edema cannot be excluded.

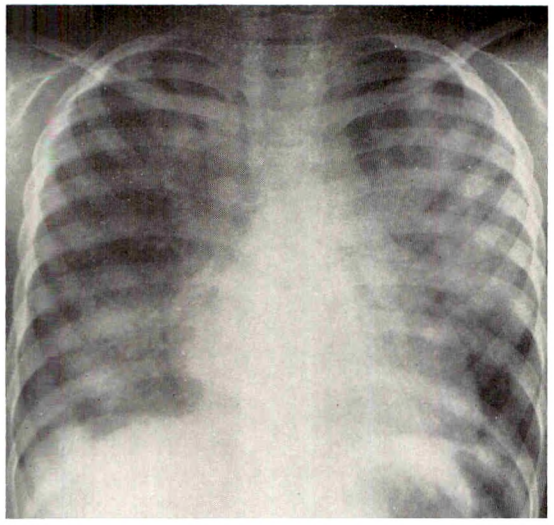


Fig. 4. March 31, 1961: Persistent cardiac enlargement and pneumonic changes in the right lower and left middle and upper lung field are demonstrated.

had a decidedly pathologic increase in gamma globulin without other symptoms of active disease. They believed that there might be an inherited tendency to overproduction of gamma globulin. The development of disseminated lupus erythematosus might then be provoked by unfavorable antigens.

Seip[12] reported an unusual case in which a woman of 29 developed signs of subacute systemic lupus erythematosus during her fourth pregnancy. In connection with delivery an exacerbation of the disease occurred with pronounced hemolytic anaemia, a positive Coombs' test, leukopenia, and a mild thrombocytopenia. Her newborn infant also developed a severe hemolytic anemia, leukopenia and thrombocytopenia, but with compete restitution at $3\frac{1}{2}$ months of age following treatment with blood transfusions and prednisone.

SUMMARY AND CONCLUSIONS

1. A case is reported which is apparently the first recorded instance in which an asymptomatic mother gave birth to a child with disseminated lupus erythematosus.

2. Roentgenographic changes included cardiac enlargement due to anemia and probable myocardial damage. Pulmonary changes included multiple episodes of pneumonia and edema with some prominent interstitial changes.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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EDITORIALS

PEDIATRIC RADIOLOGY

THE concept that infants and children are merely small-scale adults with small-scale adult diseases was recently refuted when a new radiology resident came to The Children's Hospital of Philadelphia. This doctor was no new boy fresh from internship, but one with several years of training in internal medicine, a tour of duty in the Armed Forces, and almost three years of radiology training in a large Philadelphia medical school hospital. Initially confused by the new surroundings, new techniques, different machines, and doubtless by the general noise level which accompanies the study of infants and children, nevertheless he watched attentively as the normal run of disease was going through the department. Near the end of the first week, he finally observed: "But this is a completely different specialty,—even the diseases aren't the same."

Undoubtedly part of his reaction was stimulated by being surrounded by very sick children, some with dreadful congenital anomalies, but this is not what he meant. During that week he had found that every examination must be looked at and considered from a different point of view and with a different set of standards than in adult institutions.

For example, even with a simple chest roentgenogram, he fell into the trap of diagnosing a cardiac enlargement without taking into consideration that he was looking at an expiratory roentgenogram. He had to learn that the diaphragm must be down before the apparent large heart becomes significant. Conversely, he did not know the significance of a hyperaerated chest. Week after week in a children's hospital, a baby is referred because of recurrent respiratory infections. These infants often come with several chest examinations in which the diaphragm is down to the tenth or eleventh ribs and the lungs appear clear. By comparative adult standards this would be normal, but in babies these are hyperaerated chests, and perhaps indicative of fibrocystic disease or congenital heart disease. The significance of hyperaeration and hypoaeration can only be learned by seeing as many roentgenograms of babies as a radiologist normally sees of adults.

Probably the most difficult thing to learn in radiology is a concept of normality. Once the normal is established, deviations from the normal are relatively simple to perceive. Perhaps the right name for a particular deviation will not always be apparent, but at least it will be recognized as a departure from the normal. Since adults have stopped growing, one set of standards is adequate. In growing infants and children, normal standards change month by month and year by year. The writer recalls the first lateral neck roentgenogram of a baby with a suspected foreign body that he was called upon to interpret. The anterior arch of C1 lying out there all alone in the soft tissues, with no connection to the rest of C1, obviously appeared to be a foreign body. The second such case had a long styloid process coming down into the retropharyngeal soft tissues. This too appeared to be a foreign body, because babies should not have long calcified styloid processes. Actually, some do and some do not. Almost every radiologist has called an apparent soft tissue mass in the retropharyngeal space an abscess, when in reality it was nothing more than the normal soft tissues always seen during expiration. On oblique views of the chest, the writer had to learn that the developing sternal bodies are not

hilar lymphadenopathy. Normal pulmonary vessels are identical with pneumonia when the film is exposed in expiration. How many of us have called the developing trochlea at about nine years of age a fracture of the elbow? How many radiologists constantly speak of aseptic necrosis of various bones—especially the tarsal navicular and the calcaneal epiphysis—when in reality the irregular epiphysis is a variant of normal? How many of us have talked about an abnormally dilated esophagus or an abnormally large colon in a baby, before we learned that babies grow up to their esophagus and to their colon? The list of errors could be extended to almost every bone and organ in the body, simply because the variations of normal in the growing child are so great.

Adding to the errors in diagnosis is the fact that the diseases themselves are different in children. We all learned that abdominal pain in adults is best studied by examining the gastrointestinal tract with some contrast medium. In the infant and child these studies have a very low yield of positive findings, whereas intravenous urography is the more informative examination. The tremendously high incidence of congenital anomalies of the genitourinary tract and the even higher incidence of urinary tract infections, especially in the female, make intravenous urography the most productive special study. It is only relatively recently in this country that we have learned that the intravenous urographic study is not completed until we have also visualized the urethra during the act of voiding. Indeed, both the diseases and the methods of study are different.

Many radiologists are unaware of the high incidence of cancer in children. Malignant disease is second only to accidents as the chief cause of death from ages one to fourteen. It is a different type of malignancy, however, than one finds routinely in the adult. Leukemia, in all its protean manifestations, probably heads the list. After this come the sarcomas. Carcinomas of the adult tend to give rise to symptoms early in their development, because they are epithelial tumors which frequently affect the covering or lining layers of a viscus. The sarcomas, being of connective tissue origin, may be silent until extreme growth has occurred or metastasis has taken place. For this reason, all solid and cystic tumors in childhood must be considered malignant until proven otherwise.

When the writer ceased working with adults, he thought he had put behind him the entire field of functional problems. It did not take long to discover that children are perhaps even more disturbed than adults. Children exhibit their disturbances with a myriad of organic complaints: abdominal pain, chest pain, headaches, chronic constipation, enuresis, vomiting, etc. This is important for the radiologist to know. Frequently children are referred to The Children's Hospital of Philadelphia after having had repeated roentgen examinations of every system in the body. From pediatrician to pediatrician, and therefore from radiologist to radiologist, these children have wandered, solely because of a lack of appreciation of functional disorders. These children have been exposed to much unnecessary radiation. This does not mean that roentgen examinations should not be carried out, but it does behoove the radiologist to perform a proper and definitive study in the first instance, and, if his study rules out organic disorders, to suggest that a visit to a child psychiatrist might be of greater value than further roentgen examinations.

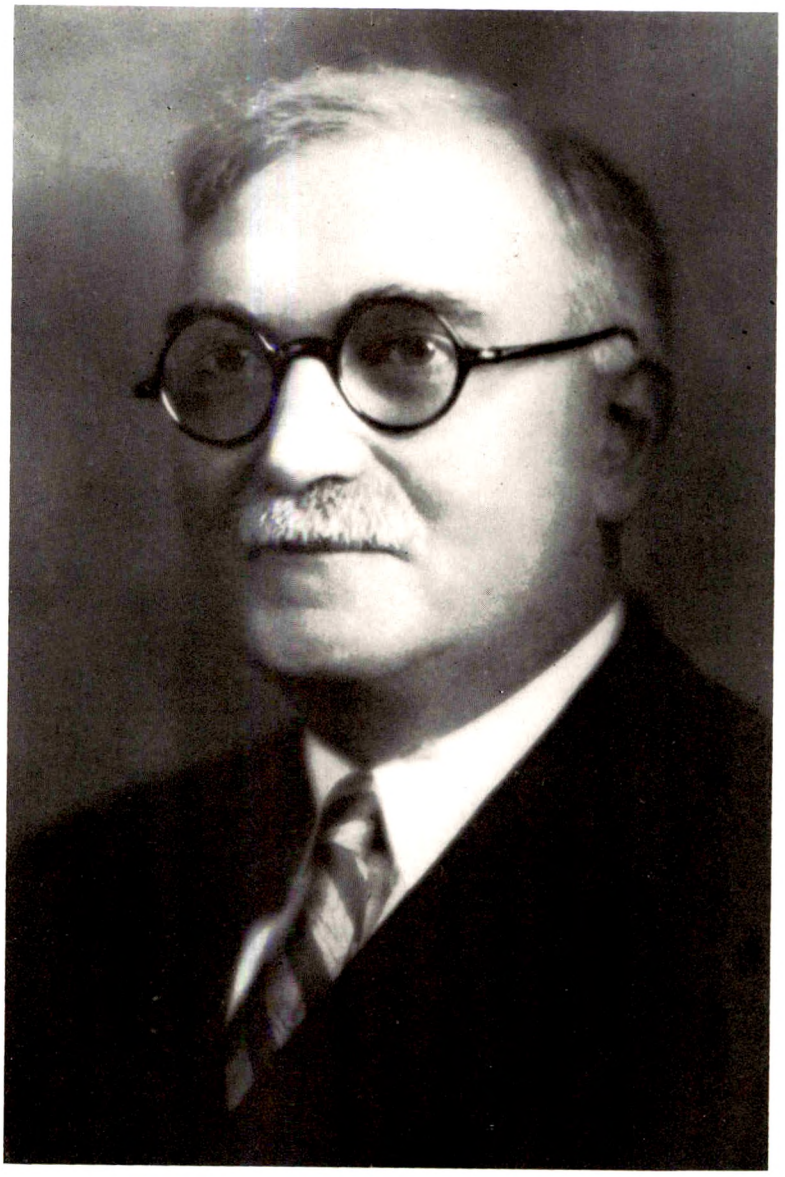
Working in a children's hospital, it is deeply disturbing to see the quality of the roentgen examination which often accompanies the referred patient—the pure black or pure white films which defy interpretation, often from hospitals where studies of adults are of the highest quality. Infants and children form the age group which, above all, should be protected against unnecessary radiation. Repeat studies produce unnecessary radiation. Would not it prevent a significant amount of radiation if radiologists paid as much scrupulous atten-

tion routinely to "little people" as is paid to adults? A repeat examination of a sixty-five year old adult is infinitely less disturbing than the repetition of a study of a baby because the quality of the original examination left the diagnosis in doubt.

These are random thoughts on the importance of Pediatric Radiology. This subspecialty has now grown so that it has its own Society numbering about 70 members. This is an encouraging development. It is also encouraging to note that more and more questions pertaining to "little people" are being asked by the American Board of Radiology of their prospective diplomates. After all the stress placed on adult radiology, it is gratifying to observe the growing attention at the other end of the line—on the proper diagnosis for the infant or child who has his whole life before him.

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LEON THEODORE LEWALD, M.D.
1874-1962

ONE of the most revered grand men of Radiology died on June 16, 1962, at the age of 88. He was Leon Theodore LeWald.

His radiologic interest spanned a period of 66 years. It began in 1896 while interning at the Knickerbocker (then the S. Hood Wright) Hospital, when he borrowed a large induction coil from Dr. W. G. LeBoutillier, a surgeon, and obtained an Edison tube from another source. Then in 1897, while an intern at the University (then the New York Post-Graduate) Hospital, he used a makeshift x-ray installation with Dr. Samuel Lloyd, a surgeon, in the exact location of the present x-ray department.

His scientific career closed with his attendance at the 1962 Annual Meeting of the American College of Radiology in New York.

Dr. LeWald was born in Port Henry, New York, on Lake Champlain, in 1874. He studied at Union College, Schenectady, New York, and received the degree of Doctor of Medicine from the College of Physicians and Surgeons of Columbia University in 1895.

While an Instructor in Pathology at New York University and Bellevue Hospital Medical College from 1897 to 1902, he performed 5,000 autopsies. This vast experience gave him a solid background for investigation and the many contributions he subsequently made to scientific medicine.

Dr. LeWald served in the Spanish American War, as Acting Assistant Surgeon, U. S. Army, and then was commissioned as Captain and Assistant Surgeon in the 71st Regiment National Guard, State of New York and, from 1902 to 1910 he was in the Armed Forces, having been commissioned by President Theodore Roosevelt as an officer in the Regular Army Medical Department. After graduation from the Army Medical School he served for several years in the Philippines, at Iloilo, and then in Guimeras, where in 1904 he discovered a hitherto unknown species of mosquito to which his name was subsequently given—Megarhinus LeWaldi.

While in the Philippines, he was shipwrecked in the China Sea and hit upon the idea of helping pass the time away by unpacking his microscope and showing his companions microscopic life in sea water. Then, in examining material from the mouths of adults and children, he found an amoeba which was present in practically all of the passengers and crew. This proved to be "Entamoeba gingivalis" which later was of great concern to health authorities because of its presence in children of the New York schools, but referring to Dr. LeWald's finding of this amoeba in healthy persons, Dr. Anna Williams of the Health Department was able to dispel this fear. After he had examined 35,000 army recruits, Captain LeWald inaugurated x-ray chest examinations, thus antedating the mass x-ray examinations now used routinely in industry, education and health examinations. He was probably the first to recommend the use of the x-ray in *routine* chest examinations in 1908.

In 1910 Dr. LeWald was made head of the x-ray laboratory at New York University Bellevue Hospital Medical School, succeeding the famous electrical engineer and physician, Eugene Caldwell, who had established this first endowed x-ray laboratory in the country as the Edward N. Gibbs X Ray Laboratory. From 1917 to 1939, Dr. LeWald was Professor of Roentgenology—one of the first in this country.

During World War I Major LeWald was the Commanding Officer of the U. S. Army School of Roentgenology at Cornell University Medical College, New York City.

Dr. LeWald was a Follow of the American College of Radiology, and was one of the first Chancellors. He earned the degree of D.M.R.E. at Cambridge, England, in 1926 and became a Diplomate of the American Board of Radiology. He reorganized the x-ray department at St. Luke's Hospital and served as Director from 1911 to 1924. In 1917 he organized the x-ray department of Willard Parker Hospital, probably the first Contagious Disease Hospital to have its own x-ray department. He was Consultant to the Vassar Bros. Hospital (Poughkeepsie, New York), Hackensack Hospital, New Jersey, and the Knickerbocker Hospital.

The literary contributions to medical science made by Dr. LeWald are many and include: *Observations on Mediterranean Anemia*, *Diverticulitis of the Colon*, *Mucocele of the Appendix*, *Congenital Absence of the Superior Wall of the Orbit*, *Findings Relating to Primary Cancer of the Lungs*, *Anatomical X-Ray Determination of the Position of the Heart Valves*, *Effects of Altitude on the Size and Position of the Heart in Aviators*, and *Transposition of the Viscera in Man*. His

studies on this latter subject showed that in World War II U. S. Army examinations revealed that this condition, namely *right-sided* heart and *leftsided* appendix and gall-bladder, was present in approximately 1 of each 10,000 persons. Dr. LeWald himself collected probably the largest series of these cases—a total of 40 personally examined—and to demonstrate the relative frequency of the condition and its importance he devised the slogan: "A transposition person is born every day in the United States at the present high rate of birth."

Early in his career, with other pioneers, he began the study of measured dosage of x-rays in medicine and one may here quote from a letter to him from Dr. William D. Coolidge, a former Director of the General Electric Research Laboratory and inventor of the present x-ray tube:

"I often think how fortunate it was that, as a result of the work of pioneers like yourself in the medical use of the x-rays, it was possible to avoid the tremendous radiation damage which would otherwise have resulted from the early work on the production of plutonium. Without the knowledge which had been gleaned through the years on the physiological effects of measured quantities of radiation, it would of course have been impossible to provide adequate protection for the large number of people employed in the big atomic plants."

Dr. LeWald was a member of the Society of Medical Jurisprudence for over 40 years. For many years he was Treasurer, Trustee, and Chairman of its Program Committee. He was elected to Honorary Membership in 1961.

Dr. LeWald was the only early member of the New York Roentgen Society, which was founded in 1912, to be alive and present during its Golden Anniversary Celebration in 1962.

M. H. POPPEL, M.D.



ROBERT A. ARENS, M.D.
1889-1962

DR. ROBERT A. ARENS, Senior Attending Physician at Michael Reese Hospital and Medical Center, died May 31, 1962, after a prolonged illness.

Dr. Arens was born in Chicago in 1889. After graduating from Crane Technical High School in 1907, he became interested in the then new field of x-ray technique and development. For the next six years he worked with various aspects of x-ray equipment, which included design, service, instruction and selling. He attended medical courses in St. Louis for two years, and in 1915 he was asked to develop the x-ray department at Michael Reese Hospital. Concurrently with his activities at Michael Reese he pursued his medical studies, and received his M.D. degree from the Chicago

Medical School in 1918. He was licensed to practice medicine in Illinois in January of 1920, and was appointed Director of the Department of Radiology, a position he held until his retirement in 1954, an association of almost 40 years.

Dr. Arens pursued his interest in the technical developments of our specialty and made many valuable contributions. He devised a hand-fluoroscope which greatly aided in the localization of foreign bodies, a radiographic table for pneumoperitoneum examinations, rack film dryers, and many other items which eventually became standard equipment. He assiduously improved the facilities of the department in line with the general development of the specialty and the continuously growing demands of an increasing hospital bed capacity.

He conducted an extensive training program for residents and stimulated and encouraged the young men working with him by his own devotion to his work. He also established a school for x-ray technicians. His advice was constantly sought by the staff members and he very actively participated in intramural conferences and seminars.

In addition to the direction of his own department, he served for many years on numerous committees of the medical staff where his organizational talents, his thoroughness, his fairness, and his judgement were highly appreciated by his colleagues. He contributed more than 60 published papers to the radiological literature and spoke before many medical societies as well as before civic groups.

He served as consultant to the Winfield Sanitarium and the Illinois Central Hospital.

In organized medicine Dr. Arens was also very active. He was a fellow of the Chicago Roentgen Society and came to serve as its secretary, vice-president and president. He was for many years a member of the governing council of the Chicago Medical Society. He served as a vice-president of the American Roentgen Ray Society in 1939. He was a member of the Radiological Society of North America, and served as a vice-president in that organization from 1937 to 1939. He was a member of the Board of Chancellors of the American College of Radiology from 1937 to 1942, and was chairman of the section of radiology of the American Medical Association from 1943 to 1945. In these national societies he served as a member and chairman of many of the subcommittees, including the scientific exhibits committee of the two major radiological societies. His organizational ability, his deep interest in the proceedings, and his attention to detail made him an almost indispensable team-member at all the meetings which were held in Chicago by national societies.

During the First World War, Dr. Arens served as Captain in the Medical Corps and maintained his reserve commission for many years. His interest in military affairs and training carried him into activities in the St. John's Military Academy of Delafield, Wisconsin, where he founded the important and influential "Dad's Club."

Throughout his life Dr. Arens' first loyalty was to Michael Reese Hospital and Medical Center, where he began and ended his successful career. He served this institution competently and faithfully. His friends and associates will best remember him for his stern character, his forcefulness, his absolute integrity and honesty, his penchant for truth, and his kindness.

Dr. Arens was a considerate and thoughtful husband and father. Our sympathy is extended to his wife Rosella, his son Robert, his daughter Margaret (Craig), and his surviving brother Carl.

The Board of Directors of Michael Reese Hospital and Medical Center has honored Dr. Arens by naming the newly built diagnostic radiology department in the B. & E. Mandel Clinic for him in recognition of his pioneering efforts, inspired leadership, and accomplishments. His old associates feel deeply the loss of a distinguished colleague, a dear friend, and an honorable man.

Erich M. Uhlmann, M.D.



MAX RITVO, M.D.
1897-1962

For now he lives in fame, though not in life.

RADIOLOGISTS the world over learned with shock and sorrow of the sudden passing of Dr. Max Ritvo on March 29, 1962.

Born in Wilno, Russia, his parents brought him to this country in 1899 when he was 2 years old. He early demonstrated the brilliance and boundless energy which characterized his life. Typical was his working as a pharmacist while attending Harvard College from which he graduated with honors. He then completed his medical studies at the Harvard Medical School and in 1923 interned at the Massachusetts

General Hospital, under Dr. George W. Holmes. This was a particularly warm memory, and he spoke with feeling of those early days of radiology in this country. After leaving the Massachusetts General Hospital, he joined the radiology staff at the Boston City Hospital which was to become his professional home. His many years at that institution culminated in 1945 with his appointment as Roentgenologist-in-Chief and Director of the Department of Radiology. With dedication, tenacity, and single-mindedness he advanced the department of his beloved "B.C.H." to its present position amongst the leading teaching centers, and attracted trainees from all over the world. Characteristically, he was deeply engaged in plans for further major improvements when the end came.

His academic career was equally illustrious. He was an assistant clinical professor of radiology at Harvard, and had teaching appointments at both Tufts Medical School and the Boston University School of Medicine. Yet this dynamo ran an extremely busy private practice as well as being senior radiologist at three other hospitals in the Boston area and consultant at several other local institutions. Still there was time for service in civic, professional, and religious organizations. He was president of the New England Roentgen Ray Society in 1954–1955, Orator of the Massachusetts Medical Society in 1959, an alternate delegate to the American Medical Association, officer in numerous professional, civic, and fraternal organizations, and a leader of his religious community. Despite these manifold demands on his time and energies, he was extraordinarily productive with more than 60 major articles appearing in the medical literature, not to mention 4 texts in roentgen diagnosis—one now in its second edition—which became standard works of reference the world over. In all these activities, he was assisted by his devoted wife, Frances, who worked with skill and efficiency to ease the demands made on him. While quietly proud of his daughter, Marion, a member of the Massachusetts Bar and his son, Dr. Edward R. Ritvo, it was his five grandchildren who were his special delight.

His associates and students will recall him as forever patient, kind, and attentive, always ready to help with his enormous experience and diagnostic prowess.

This extraordinary man excelled in all he undertook. Efficient administrator and executive, able medical statesman, brilliant scholar, wise and compassionate physician—he was all these and many more.

He will be missed.

G. J. D'Angio, M.D.

NEWS ITEMS

THE AMERICAN BOARD OF RADIOLOGY

The Spring 1963 examination will be held at the Sheraton Hotel, Philadelphia, Pennsylvania, June 3–7, inclusive. Deadline for filing applications is December 31, 1962. There will be a Special Examination in Nuclear Medicine for diplomates in Radiology or Therapeutic Radiology if sufficient applications are received. The Fall 1963 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, December 2–6, inclusive. Deadline for filing applications is June 30, 1963.

The following announcement is also made:

Pursuant to the policy of the American Board of Radiology of continuously elevating the educational value of training programs in Radiology and of improving all standards pertaining thereto, the Board now has under advisement the establishment of programs which will permit departments with certain qualifications to be approved for the training of residents in Diagnostic Radiology alone or Therapeutic Radiology alone. The required training period in the chosen field will be three years.

It will be possible for a particular department to be approved for:

a. Radiology
b. Diagnostic Radiology
c. Therapeutic Radiology

A department presently approved in the full field of Radiology may then request approval in either one or both of the subdivided fields. A department not qualifying for approval in the entire field may seek recognition in either Diagnostic Radiology or Therapeutic Radiology.

Qualifications for approval in the divided programs are anticipated to be at a high level. A high staff-resident ratio, a broad scope of available material for resident teaching and a general hospital which provides all aspects of a superior educational program are expected to be minimal requirements. Thorough evaluations by the American Board of Radiology and the Council on Medical Education and Hospitals of the American Medical Association will be conducted.

There are many details to be worked out. The exact date of implementation of these new programs is therefore indefinite. Due notice will be given when applications can be received, but this program cannot be implemented until cleared by the House of Delegates of the American Medical Association.

FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND

At the Royal College of Surgeons in Ireland on June 22, 1962, the President of the Royal College of Surgeons in Ireland, Mr. N. Kinnear, F.R.C.S.I., conferred the Fellowship of the Faculty of Radiologists on 45 radiologists, among whom were Dr. Bernard J. O'Loughlin of Los Angeles, California, and Dr. Charles C. Verstanding of New Haven, Connecticut.

On June 23, 1962 the Honorary Fellowship was conferred on Dr. Ernest Rohan Williams, London, England; Dr. Peter James Kerley, London, England; and Sir Frank Percivale Montgomery, Belfast, Northern Ireland.

Following the ceremony the First Annual Dinner of the Faculty of Radiologists, Royal College of Surgeons in Ireland, was held in the College Hall. The Dean of the Faculty of Radiologists, Mr. D. J. Riordan, F.R.C.S.I., F.F.R.R.C.S.I., during his address, welcomed all the new Fellows and guests. He thanked the members of the Board of Faculty of Radiologists, especially the Vice-Dean, Dr. S. J. Boland and the Honorary Secretary, Dr. F. H. Cross for their untiring work during the past year. He spoke of the future of the new Faculty

of Radiologists which under the auspices of the President and Council of the College propose to draw up a formal course of instruction for medical graduates who aspire towards the Fellowship. The syllabus will include set courses in Diagnostic Radiology, Radiotherapy and Nuclear Medicine and specific examinations for the Fellowship will be held in these subjects. Courses and examinations for the Diploma in Radiology and Nuclear Medicine will also be held.

The Registrar is Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., St. Stephens Green, Dublin 2, Ireland.

CLINICAL CONFERENCE ON TUMORS OF THE SKIN

"Tumors of the Skin" will be the subject of the Clinical Conference to be held at the University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas, November 9 and 10, 1962. Papers will be presented by 20 scientists in the fields of etiology, epidemiology, diagnosis and treatment of cancer of the skin. This is the seventh in a series of Annual Clinical Conferences at M. D. Anderson Hospital.

The session on etiology and epidemiology of tumors of the skin will be presided over by J. Leslie Smith, Jr.; the session on diagnosis by William S. MacComb; and the session on treatment by Gilbert H. Fletcher.

The proceedings of this conference will be published in a monograph entitled "Tumors of the Skin."

COURSE IN INSTRUMENTAL AND CHEMICAL TECHNIQUES OF ACTIVATION ANALYSIS

A course in the "Instrumental and Chemical Techniques of Activation Analysis" will be presented by the ORINS Special Training Division, September 24 to October 5, 1962.

This special course is designed for the scientist or engineer who has already done some work involving radioisotope techniques and is now interested in applications involving neutron-activation analysis. It will include both lecture and laboratory work covering such pertinent topics as gamma spectroscopy, multichannel analyzers, neutron sources, computer applications, and radiochemical separations.

Prominently featured in the course will be the Special Training Division's new neutron generator—a source capable of producing up to 10^{10} neutrons per second. The device will permit experiments in flux determination, the use of fast or slow neutrons, and analyses of the spectra of extremely short-lived radioisotopes.

Under its contract with the U. S. Atomic Energy Commission, the Special Training Division of ORINS—a nonprofit educational corporation of 38 universities—has provided radioisotope-technique training to more than 4,800 scientists from all 50 states and 58 foreign countries.

Further information about the special course in activation analysis can be obtained by writing Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tennessee.

LECTURES ON LESIONS OF THE GASTROINTESTINAL TRACT IN MEXICO CITY

The Departamento de Investigación y Enseñanza científica and the X-Ray Department of the Mexico City General Hospital have invited Dr. Richard H. Marshak to give a series of lectures on lesions of the gastrointestinal tract on October 11, 12 and 13, 1962.

The organizers invite all radiologists who might be interested to attend these lectures and at the same time to visit Mexico. The registration fee is 200 pesos (16.00 U.S.A. dollars).

Further information may be obtained from: Dr. Aquilino Villanueva, Chairman, Departamento de Investigación y Enseñanza científica del Hospital General, Dr. Balmis No. 148, México 7, D.F., México.

BOOKS RECEIVED

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

THE INTERPRETATION OF TOMOGRAMS OF THE HEAD; AN ATLAS. By Marvin L. Daves, M.D., Assistant Professor, Department of Radiology, University of Colorado Medical Center, Denver, Colorado; and William E. Loechel, Director of Medical Illustration, Henry Ford Hospital, Detroit, Michigan. Cloth. Pp. 248, with 172 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1962.

THE GYNECOLOGIC PATIENT; A PSYCHO-ENDOCRINE STUDY. By Somers H. Sturgis, M.D. Cloth. Price, $7.75. Pp. 243, with 18 charts. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, N. Y., 1962.

TREATMENT OF CANCER AND ALLIED DISEASES. Second edition. VOLUME VI. TUMORS OF THE FEMALE GENITALIA. Edited by George T. Pack, M.D., F.A.C.S. Associate Professor of Clinical Surgery, Cornell University Medical College; Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Surgeon, Pack Medical Group, New York, New York; and Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery, Attending Surgeon, and Chief of the Neoplastic Service, Department of Surgery, New York Medical College, Flower and Fifth Avenue Hospitals; Attending Surgeon, Hospital for Joint Diseases; Surgeon, Pack Medical Group, New York, New York. Cloth. Price, $16.50. Pp. 381, with 272 illustrations of which 10 are in color. Hoeber Medical Division, 49 East 33rd Street, New York, N. Y., 1962.

LUMBAR DISKOGRAPHY AND ITS CLINICAL EVALUATION. By Dr. L. Walk, Central Hospital, Eskilstuna, Sweden. Cloth. Price, $8.50. Pp. 135, with 41 illustrations and 11 tables. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1962.

PROGRESS IN RADIATION THERAPY. VOL. II. Edited by Franz Buschke, M.D., Professor of Radiology, University of California School of Medicine, San Francisco, California. Cloth. Price, $12.50. Pp. 266, with illustrations. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, N. Y., 1962.

RADIATION CHEMISTRY OF POLYMERIC SYSTEMS. By Adolphe Chapiro, Laboratoire de Chimie Physique de la Faculté des Sciences de Paris, Paris, France. Cloth. Price, $21.00. Pp. 712, with many illustrations. John Wiley & Sons, Interscience Division, 440 Park Avenue South, New York 16, N. Y., 1962.

ACTA RADIOLOGICA—SUPPLEMENTUM 212. PELVIC INSTABILITY IN NEWBORNS WITH SPECIAL REFERENCE TO CONGENITAL DISLOCATION OF THE HIP AND HORMONAL FACTORS; A ROENTGENOLOGIC STUDY. By Dr. Lars Andrén. Paper. Pp. 66, with 34 figures. Acta Radiologica, Stockholm 2, Sweden, 1962.

FLUORIDE IN CLINICAL MEDICINE. By George L. Waldbott, M.D., 2930 West Grand Boulevard, Detroit 2, Michigan. Paper. Price, $3.50. Pp. 60, with 15 figures and 10 tables. S. Karger, Arnold-Böcklinstr. 23–25, Basel, Switzerland, 1962. In the U.S.A.: Albert J. Phiebig, P. O. Box 352, White Plains, N. Y.

LA FLEBOGRAFIA EPATICA. By L. Belli, D. Galmarini, and U. Di Francesco, Milan, Italy. Cloth. Pp. 221, with 137 illustrations. Minerva Medica, 1962.

CHIRURGIE DU SYSTÈME DU CANAL HÉPATIQUE; LÉSIONS BENIGNES. By P. Mirizzi, Professeur honoraire à l'Université de Córdoba, Argentina. Paper. Pp. 203, with 115 illustrations. Masson & Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris (VIe), France, 1962.

LA TÉLÉCOBALTHÉRAPIE; PRINCIPES—DOSIMÉTRIE, PRINCIPALES APPLICATIONS EN CANCEROLOGIE. By H. Fourquier, Chef de Service de Physiothérapie du Centre régional anticancéreux de Montpellier. Paper. Pp. 240, with 131 figures. Masson & Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris (VIe), France, 1962.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Shoreham Hotel, Washington, D. C., Oct. 2–5, 1962.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Justin J. Stein U.C.L.A. Medical Center, Los Angeles 24, Calif. Annual meeting: Mark Hopkins Hotel, San Francisco, Calif., April 1–4, 1963.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb.
Treasurer, Dwight Vincent Needham, 713 E. Genessee St., Syracuse, N. Y. Annual meeting: Palmer House, Chicago, Nov. 25–30, 1962.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 6–9, 1963.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: Atlantic City, N. J., June 16–20, 1963.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. The Fall 1962 examination will be held at the Pioneer Hotel, Tucson, Arizona, the first week in December; the deadline for filing applications was July 1, 1962. Please note that at this session Nuclear Medicine will become a mandatory part of the examination. All candidates, excluding re-examinees, who are applying for examination in Radiology or Therapeutic Radiology for the December, 1962 session or thereafter *must submit a Nuclear Medicine application* in addition to their basic application. This applies even to those applicants whose basic applications are on file but who will not yet have appeared for examination. The Spring 1963 examination will be held at the Sheraton Hotel, Philadelphia, Pennsylvania, June 3–7, inclusive. Deadline for filing applications is December 31, 1962. There will be a Special Examination in Nuclear Medicine for diplomates in Radiology or Therapeutic Radiology if sufficient applications are received. The Fall 1963 examination will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, December 2–6, inclusive. Deadline for filing applications is June 30, 1963.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

TENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

EIGHTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colo. Meeting to be announced.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY
Treasurer, Raymond Maxson, 86 E. Randolph St., Chicago, Ill.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. E. Lawrence Ganter, 1820 E. Polk St., Phoenix 34, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary, Dr. John A. Campbell, Indiana University Medical Center, Indianapolis, Ind. Annual meeting: Yale University Medical Center, New Haven, Conn., May 24–25, 1963.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bernard J. Ostrum, 2412 North 52nd St., Philadelphia, Pa.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary, Dr. Leslie L. Alexander, 257 New York Ave., Brooklyn 16, N. Y. Meets first Thursday of each month October through May.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Edward A. Dunlap, Jr., 35 Danbury Lane, Kenmore 17, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Edward W. Carsky, Crouse-Irving Hospital, 820 S. Crouse Ave., Syracuse, N. Y. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Dana R. Schmidt, 1500 West Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Abram Cannon, Chicago Wesley Memorial Hospital, 250 E. Superior St., Chicago 11, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Ward D. Heinrich, Huron Road Hospital, Cleveland 12, Ohio. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. Seward Imes, 1845 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. James L. Krieger, 85 Jefferson St., Hartford 6, Conn. Meets first Friday in February and April.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary, Dr. Robert D. Moreton, 816 Medical Arts Bldg., Fort Worth, Tex. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. Arch H. Hall, Harper Hospital, Detroit 1,

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland, Calif.

East Tennessee Radiological Society
Secretary, Dr. J. Marsh Frere, Jr., 205 Medical Arts Bldg., Knoxville, Tenn. Meets in January and September.

Eastern Conference of Radiology
Secretary, Arrangements Committee, Dr. Bernard S. Wolf, The Mount Sinai Hospital, 11 E. 100 Street, New York 29, N. Y. Annual meeting to be announced.

Eastern Radiological Society
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

Florida Radiological Society
Secretary, Dr. Richard D. Shapiro, 1680 Meridian Ave., Miami Beach, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1800 Druid Rd., Clearwater, Fla.

Georgia Radiological Society
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. T. Hunter Pryor, Jackson Memorial Hospital, Miami 36, Fla. Meets monthly, third Wednesday, at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary, Dr. Harry I. Berland, St. Louis, Mo.

Houston Radiological Society
Secretary, Dr. W. E. Kirkpatrick, 2708 Weslayan, Houston 27, Tex. Meets last Monday each month in fall winter and spring, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

Illinois Radiological Society
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

Indiana Roentgen Society, Inc.
Secretary, Dr. Richard A. Silver, 712 Hume Mansur Bldg., Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Roger K. Wallace, Riley County Hospital, Manhattan, Kan. Meets in spring with State Medical Society and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Lawrence A. Davis, 226 East Chestnut St., Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. Abraham Berens, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Los Angeles Radiological Society
Secretary, Dr. Saul Heiser, Los Angeles, Calif. Meets second Wednesday of month in September, November, January, March and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Charles W. Capron, Maine Medical Center, Portland, Me. Meets in June, September, December and April.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. Irving K. Ettman, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday of each month September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Abraham Marck, Mayfair Professional Bldg., Milwaukee 13, Wis. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society
Secretary, Dr. Frank J. Anderson, 810 E. 27th St., Minneapolis 7, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Jack K. Goodrich, University Medical Center, Jackson, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. John M. Fritts, 1224 S. Higgins Ave., Missoula, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Herbert Kantor, 71 S. Oyster Bay Rd., Syosset, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska State Radiological Society
Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society
Secretary, Dr. George F. Fraser, Las Vegas, Nev.

New England Roentgen Ray Society
Secretary, Dr. Robert E. Wise, 605 Commonwealth Ave., Boston 15, Mass. Meets third Friday of each month, October through April, at The Longwood Towers, Brookline, Mass. at 4:30 P.M.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. James J. Nickson, Memorial Hospital, New York, N. Y. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. A. B. Croom, 624 Quaker Lane, High Point, N. C. Meets in the spring and fall each year.

North Dakota Radiological Society
Secretary, Dr. John Jestadt, Depuy-Sorkness Clinic, Jamestown, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Lester I. Citrin, St. Mary's Hospital, Troy, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Northwestern Ohio Radiological Society
Secretary, Dr. Anthony F. Lalli, 421 Michigan St., Toledo, Ohio.

Ohio State Radiological Society
Secretary, Dr. Chapin Hawley, 927 Carew Tower, Cincinnati 2, Ohio. Annual Meeting: Dayton, Ohio, May, 1963.

Oklahoma State Radiological Society
Secretary, Dr. Simon Pollack, Utica Square Medical Center, Tulsa, Okla. Meets in January, May and October.

Oregon Radiological Society
Secretary, Dr. James A. Schneider, St. Vincent Hospital, Portland 10, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. John N. Burkey, 555 Dental Bldg., Seattle, Wash. Annual meeting: Seattle, Wash.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Frederick R. Gilmore, Clearfield Hospital, Clearfield, Pa. Annual meeting: Bedford Springs Hotel, May 23–25, 1963.

Philadelphia Roentgen Ray Society
Secretary, Dr. Antolin Raventos, 3400 Spruce St., Philadelphia 4, Pa. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Ross H. Smith, St. Margaret Memorial Hospital, Forty-Sixth St., Pittsburgh 1, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. William C. Duffey, Cincinnati, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Major Michael A. Lopiano, 784 Hoomalimali St., Pearl City, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Andrew F. Giesen, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Robert Scanlan, St. Vincent's Hospital, Los Angeles, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Harry J. Pinsky, 386 Monroe Ave., Rochester 7, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 16–18, 1962.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Charles G. Campbell, 6673 Avenida Manana, La Jolla, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. Walter Coulson, San Francisco General Hospital, San Francisco 8, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Wayne P. Whitcomb, Hospital of St. Raphael, New Haven, Conn. Meetings are held bimonthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. Martin A. Thomas, 1150 Connecticut Ave., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. Robert D. Sloan, University Medical Center, 2500 North State Street, Jackson 6, Miss. Annual meeting: Hotel Fontainebleu, Miami Beach, Fla., Nov. 12–15, 1962.

Section on Radiology, Southern Medical Association
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Section on Radiology, Texas Medical Association
Secretary, Dr. Frederick J. Bonte, University of Texas Southwestern Medical School, Dallas 35, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. Richard G. Lester, Box 151, Medical College Station, Richmond, Va. Annual meeting: Shoreham Hotel, Washington, D. C., Oct. 1, 1962.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. *Administrator*, Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill.

South Bay Radiological Society
Secretary, Northern Section: Dr. John S. Wilson, 1515 Trousdale Dr., Burlingame, Calif.; Southern Section: Dr. A. C. Mitchell, 728 Cass, Monterey, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting: Grand Hotel, Point Clear, Ala., Feb. 1–3, 1963.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Tex. Annual meeting: San Antonio, Tex., Jan. 25 and 26, 1963.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Welborn Clinic, 420 Cherry St., Evansville, Ind. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.

Virginia Radiological Society
Secretary, Dr. Powell G. Dillard, Jr., 715 Church Street, Lynchburg, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Robert H. Rosenberg, 7011 38th Ave., N.E., Seattle 15, Wash. Meets quarterly.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broaddus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Edward T. Bello, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2–37, Zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vaga Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15. México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César E. Rosa-Perez, Fondo del Segura del Estado, Parada 1, San Juan 8, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Edmonton and District Radiological Society
Secretary, Dr. S. C. Windle, 105 Northgate Bldg., Edmonton, Alberta, Canada. Meets first Tuesday of each month, October to May.

Faculty of Radiologists
Honorary Secretary, Dr. C. J. Hodson, 47 Lincoln's Inn Fields, London, W.C.2, England.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. D. J. Sieniewicz, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Que., Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. Wallace M. Roy, St. Joseph's Hospital, 30 The Queensway, Toronto 3, Ont., Canada. Meets second Monday of each month September through May.

College of Radiologists of Australia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Socété du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut., Lagenbeckstr. 1, Mainz, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

Indonesian Radiological Society
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah 1, Djakarta, Indonesia.

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Neck and Chest

Thurn, P., Schaede, A., Hilger, H. H., and Düx, A. Graduelle Beurteilung der Mitralklappeninsuffizienz im selektiven Laevokardiogramm. (Grading of mitral insufficiency in selective levocardiogram.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1962, *96*, 37–60. (Address: Prof. Dr. P. Thurn, Med. Univ.-Klinik, Bonn, Venusberg, Germany.)

Grading of mitral insufficiency is important as it helps the surgeon to plan a specific type of operation. In mitral stenosis with slight insufficiency only valvulotomy is indicated; otherwise, valvuloplasty with extracorporeal circulation is the method of choice. The most dependable procedure to determine the degree of mitral insufficiency is selective levocardiography.

Underlying changes of the mitral valve are as follows: (1) reduced valvular surface from scarring and shrinkage, (2) enlarged opening due to dilatation of the mitral ring, and (3) incomplete closure of valvular margins in ventricular systole due to shortening of chordae and papillary muscles, the dorsal cusp and the right papillary muscle being most vulnerable. A combination of all three conditions is also possible.

The authors evaluate 27 mitral insufficiencies, especially with the aid of lateral views. Twenty-four were selective retrograde levocardiograms and 3 were aortograms. The important finding was reflux of contrast material into the left atrium after it had been injected into the left ventricle via a Seldinger catheter. This reflux was rare in normal cases. In 74 normals it occurred only faintly 6 times and was caused by factors like extrasystole, tachy- and bradycardia, and asystole. It happened during diastole and faded quickly, while in mitral insufficiency it was only seen in systole. It could be avoided by aimed selective injection into the outflow tract of the left ventricle. In mitral stenosis the reflux was always absent even with extrasystole, tachy- and bradycardia and asystole.

The grading of mitral insufficiency can be judged from the following observations: (1) density and extent of reflux, (2) comparative timing of reflux and filling of the aorta, (3) emptying time of left atrium and left ventricle according to the number of systoles, (4) degree of left ventricular dilatation, and (5) entering of the catheter into the left atrium during ventricular systole.

In Grade I mitral insufficiency staining in the left atrium is incomplete, as it centers around the valve, and is less dense than in the left ventricle. In Grade II, III and IV there is diffuse staining of the left atrium. It persists for several seconds and is equally as dense as in the left ventricle. When contrast material appears first in the left atrium and only later in the aorta, a high-grade mitral insufficiency can be concluded.

The speed of emptying can be estimated only by the number of systoles and not by the actual time, as tachycardia, bradycardia, atrial fibrillation and myocardial changes play an important role. Simultaneous electrocardiographic monitoring is essential for conclusions. Marked delay of emptying suggests high-grade functional insufficiency. More rapid emptying in spite of severe anatomic changes is also possible, when the myocardium is intact and the sinus rhythm is normal.

The degree of ventricular dilatation and hypertrophy is important for proper grading, especially in combined mitral lesions. A small left ventricle in diastole suggests predominance of a stenotic component. The size of the left ventricle also depends on the degree of associated myocardial damage. The width of the coronary arteries may give an additional clue. In pure mitral stenosis, the right coronary artery is dilated and in mitral insufficiency the left.

Entering of the catheter into the left atrium was seen in 3 cases. It suggested a high degree of mitral insufficiency. Retrograde filling of pulmonary veins was observed in 3 cases, all of which belonged to Grade IV.

In addition to the functional evaluation, the selective levocardiogram also depicts localization of the valvular defects. One can see reflux in the posteromedial and anterolateral commissure and even shrinkage of the individual leaflets.—*Ernest Kraft, M.D.*

Zaky, H. A., El-Heneidy, A. R., and Foda M. T. Haemodynamic shunts in schistosomal cor pulmonale. *Brit. M. J.*, Feb., 1962, *I*, 367–369. (From: Department of Chest Disease, University of Alexandria, Alexandria, Egypt.)

Schistosomiasis is an endemic disease in Egypt and a variety of visceral manifestations secondary to invasion by the worms and ova are seen. Hepatosplenomegaly secondary to obstruction of the portal vein by worms and ova may develop. The ova may embolize the lungs and lodge in the pulmonary arterioles producing cor pulmonale. The authors note that involvement of the portal and pulmonary circulations gives rise to circulatory changes, and the hemodynamic shunts associated with these are of some interest.

Three patients with schistosomal cor pulmonale were studied by (1) right heart catheterization with multiple samples of blood; (2) dye dilution technique with injection of Evans blue dye into the aorta and sampling from the pulmonary artery to demonstrate aorto-pulmonary shunt via the bronchial arteries; and (3) aortic angiography with a balloon catheter to visualize the bronchial artery and the pulmonary artery. Their studies proved the presence of broncho-

pulmonary arterial shunts and splenic shunts. They note that schistosomiasis, when complicated by hepatosplenomegaly and cor pulmonale shows features of the Eisenmenger type of congenital heart disease.—*Richard E. Kinzer, M.D.*

Fred, Herbert L., Castle, C. Hilmon, and Cancilla, Pasquale A. Venous stars in mediastinal disease. *A.M.A. Arch. Int. Med.*, Mar., 1962, *109*, 290–296. (From: Department of Internal Medicine, Baylor University College of Medicine, Texas Medical Center, Houston 25, Tex.)

Venous stars are small dilated superficial cutaneous veins which often develop, when the venous pressure is elevated. They are most commonly seen on the lower extremities in people with varicose veins. When they appear suddenly about the chest they may indicate a recent occlusion of a major mediastinal vein.

Occasionally venous stars may be confused with arterial spiders. However, they do not pulsate, are generally dark in color, fill from the periphery and often but not always are visualized by infrared photography. In contrast, arterial spiders pulsate, are red, fill from the center and cannot be demonstrated by infrared technique.

Two case reports are presented in which the stars were the first objective manifestation of obstruction to the great thoracic veins. Both patients had bronchogenic carcinoma with obstruction of the superior vena cava in one and the left innominate vein in the other.—*Francis P. Shea, M.D.*

Cugini, A., and Sosso, A. Il megaesofago ed alcuni suoi problemi clinico-radiologici. (Megaesophagus and some of its clinico-radiologic problems.) *Ann. radiol. diag.*, 1961, *34*, 411–430. (From: Istituto di Radiologia dell'Università, Torino, Italy.)

Megaesophagus may be either congenital or acquired. It is considered to be congenital when discovered shortly after birth or if other anomalies of the gastrointestinal tract are present. It is more difficult to establish the etiology in the acquired form due to either functional or organic causes. In some cases it may be impossible to determine the underlying cause.

The various theories on megaesophagus advanced by numerous authorities are presented in the introduction. The symptoms of dysphagia, regurgitation, emesis and aspiration can easily direct one's attention to esophageal disturbance. It is not rare that the presenting symptoms are respiratory, complicating the esophageal disease.

Next the authors describe the findings on routine chest roentgenograms which direct our attention to enlargement of the esophagus and the studies with barium meal to confirm this impression. Two points are stressed in reference to the barium meal examination: (1) not only the increased size but the redundancy of the esophagus, which because of its pressure on surrounding mediastinal structures especially the branches of the right bronchus, may be responsible for the respiratory symptoms; and (2) the action of the cardio-esophageal mechanism. Both intrinsic and extrinsic lesions of the cardiac orifice, as well as the degree of atony of the esophageal musculature, can be detected by the configuration and pliability of this part of the esophagus during passage of barium.

The esophagus is fixed at three levels: the cricopharyngeal portion, the aortic area, and the diaphragmatic attachment. Thus the redundancy of the esophagus may develop above the level of the aorta in the superior mediastinum. More often it occurs between the aorta and the diaphragm. It is usually this lower segment, which is elongated and dilated, that will press on the right pulmonary tree and becomes responsible for the respiratory symptoms observed.

The article contains many fine illustrations from cases which have come under the authors' observation to emphasize the pertinent points.—*Peter E. Russo, M.D.*

Abdomen

Van Driessche, R. Congenital hypertrophy of the pylorus. *J. belge de radiol.*, 1961, *44*, 609–626. (From: City Hospital, St-Niklaas-Waas, Netherlands.)

The author discusses the relative merit and the necessity of roentgen diagnosis of congenital hypertrophy of the pylorus with evaluation of the radiation hazards in infancy. Variations in techniques and dosimetric data related to the techniques in mr per second and per minute are considered.

In some countries roentgenography is thought to be superfluous and harmful and in other areas this diagnostic aid is believed to be of value if the tumor is not palpable. The diagnosis of hypertrophic pyloric stenosis clinically becomes more certain when the clinical triad of vomiting, visible peristalsis and palpable tumor is present. Since all cases do not exhibit this classic picture it is important that the diagnosis should not be delayed until it appears and until a rather strong baby becomes a markedly undernourished one.

In order to decrease the dose of radiation to the patient the author employs exclusively a 5 inch image intensifier, operated at 0.5 ma. and 65 kvp., and a tilting board. Fluoroscopy and 6 spot films result in a dosage of 0.08 mr to the male gonads, which is considerably less than that resulting from conventional roentgenography. The tilting board consists of a small board of about 60 by 20 cm. resting on a triangular plastic prism on one end and on the table top at the other end. The baby is placed on the board with the right side against the board and the head toward the end of the board resting on the table top.

The table is tilted to an angle of about 60° so that the longitudinal axis of the patient approaches but is not quite horizontal in order to keep the barium at the bottom of the stomach. The angle of incidence is regulated by sliding the plastic prism. The typical roentgen appearance of pyloric stenosis of a long narrow canal can be visualized more readily and more definitely by an incident beam directed from the upper right to the lower left with the baby lying on the right side. The distance of the tube target to the male gonads is increased by using the tilting board.

In pyloric stenosis the length of the visible canal is not necessarily proportionate to the gravity of the clinical image. The signs of stenosis appear only when prepyloric and intrapyloric swelling of the mucous membranes and spasticity are present. The length of the canal characterized the extent of the hypertrophy, the retention of the barium the gravity of the spasm.—*J. N. Ané, M.D.*

ZATERKA, SCHLIOMA, BETTARELLO, AGOSTINHO, MEIRELES FILHO, JOSÉ DE SOUZA, MONTENEGRO, MÁRIO RUBENS, PONTES, JOSÉ FERNANDES. Giant gastric ulcer: report of 27 cases. *Am. J. Digest. Dis.*, Mar., 1962, *7*, 236–249. (Address: Caixa postal 6209, São Paulo, Brazil.)

The size of an ulcer of the stomach has been said by some writers to be one of the criteria suggesting potential malignancy. Giant ulcer is defined as having a diameter of 3 or more cm.

The purpose of this report is to describe 27 cases of giant ulcers. Each of the 27 patients had one or more roentgenologic examinations of the stomach. Sixteen were examined by gastroscopy, and in 13, the lesion was visualized. In 9, gastric cytologic characteristics were studied.

If after 4 weeks the ulcer had not decreased in size or if after 8 weeks it had not disappeared, a diagnosis of suspect malignancy was made, and operation was recommended. Only those patients with serious heart failure or other contraindications and those who refused surgery were not operated upon.

In 8 patients the diagnosis of malignancy was made *ab initio*, and 6 were operated on. Of these, 2 had malignant lesions and 4 had benign gastric ulcers. The remaining 2 patients were not operated upon, due to medical contraindications.

Eighteen patients were submitted to the therapeutic test. In 5 disappearance of the lesion was recorded after 8 weeks. Symptoms, however, reappeared in 3, and after a second period of treatment only 1 patient showed healing. The other 2 patients were operated upon; 1 had a lymphosarcoma and the other a benign ulcer. Of the 13 patients in whom the lesion did not disappear after the therapeutic test, 7 were operated upon. Of these, 5 had benign gastric ulcers, 1 had a gastric cancer, and 1 a degenerated gastric ulcer, diagnosed according to the criteria proposed by Newcomb. The 6 remaining patients did not undergo operation for various reasons; and in 4 of the latter, the lesion disappeared after 3, 4, 5 and 13 months of treatment. Of the 2 patients in whom the ulcer did not disappear, one could not be operated upon because of a formal contraindication.

All patients with malignant lesions were males. Of the 20 patients with benign ulcers, 8 were females. The youngest patient with a malignant lesion was 34 and the oldest one was 85.

In patients with malignant lesions, the symptoms lasted between 2 months and 4 years, with an average of 2 years. In those with benign gastric ulcers, the symptoms lasted from a few days to 40 years, with an average of 7 years and 4 months.

The pattern of the epigastric pain was similar in patients with malignant and benign lesions.

One of the 7 malignant lesions and 8 of the 20 benign lesions measured more than 4 cm. in diameter; all the malignant lesions were located on the lesser curvature as were 18 of the 20 benign ulcers.

Of the 27 patients reported, 4 presented with so-called pseudotumoral gastric ulcers (those with roentgenologic, gastroscopic, and macroscopic appearances of large lesions resembling malignancy, which however on histologic study contain no neoplastic cells, neither in the ulcer nor in the associated lymph nodes). The 4 cases are described in detail.

These case reports demonstrate clearly that it is not safe to rely on size alone as a criterion of the benign or malignant nature of an ulcerated gastric lesion. They furthermore illustrate that the gross appearance of the stomach may be misleading; indeed, the perivisceritis, the reactive lymphadenitis, and the size of the lesion in these patients led the surgeon to perform total gastrectomy in 2 cases and a palliative operation in a third.

Ulcers such as these, the gross features of which resemble those of malignancy, were called "pseudotumoral" by Ledoux-Lebard. They can simulate neoplasia rœntgenologically and gastroscopically and even in a careful gross examination may be confused with cancer. In fact, in 1 case even cytologic study gave a false positive result.

Hemorrhage was the only serious complication; it appeared in 5 patients. In 1 an operation was necessary.

The therapeutic test enables one to establish the correct diagnosis of giant ulcer without histologic study. However, in the instance of pseudotumoral ulcers, the correct diagnosis can be made only on the basis of microscopic study.—*Stephen N. Tager, M.D.*

JACKMAN, M., and MIDDLEMISS, J. H. Retrograde jejunogastric intussusception: a review of 10 cases diagnosed radiologically. *Brit. J. Surg.*, Nov., 1961, *44*, 260–265. (From: The Radiodiagnostic Department, United Bristol Hospitals, England.)

Retrograde intussusception was described by Hunter in 1793; Akehurst collected 103 cases from the literature. Bozzi in 1914 first described retrograde jejunogastric intussusception occurring after surgery. Up to the present time about 130 cases have been reported, with 40 confirmed roentgenologically.

The authors present 10 cases of postoperative retrograde jejunogastric intussusception observed and diagnosed roentgenologically at United Bristol Hospitals during a 10 year period. The roentgenologic appearances are well illustrated and described. It is speculated that this condition occurs much more frequently than the literature would indicate. Plain roentgenograms of the abdomen may show gaseous distention of the stomach and afferent loop. The barium filled stomach may show a crescent shaped filling defect in which the mucosal pattern of the jejunum may be recognized. The majority of cases reduce spontaneously. Efferent loop intussusception seems more common than afferent loop intussusception, but it may be difficult to differentiate.

The authors recommend that examination be carried out in prone and supine positions since the erect position may partially reduce the lesion and cause the diagnosis to be missed. They suggest dividing the lesion into three main types: acute postoperative, acute delayed, and chronic recurrent intussusception.—*Richard E. Kinzer, M.D.*

Wong, W. T. Intestinal obstruction in children due to ascaris simulating intussusception. *Brit. J. Surg.*, Nov., 1961, *44*, 300–302. (From: Surgical Professorial Unit, Queen Mary Hospital, Hong Kong.)

In tropical regions infestation with ascaris especially in children is said to remain quite common. The author reports 4 cases of intestinal obstruction simulating intussusception which was caused by masses of round worms. Roentgenographically one sees radiolucent lines in a somewhat parallel arrangement within a more dense shadow, which can be recognized as round worms if this possibility is considered. Clinically, the obstructions occurred in the terminal ileum and on palpation the sausage shaped masses closely mimicked intussusception. However, there was a new sign—crepitation in a tympanitic mass—which should help make the diagnosis more certain. The palpable masses were located more centrally than the common ileocolic type of intussusception and there was never blood in the stool.

The treatment is discussed, but this depends on correct, firm diagnosis. The author notes that the correct diagnosis was made in his third and fourth cases.—*Richard E. Kinzer, M.D.*

Snodgrass, Richard W., and Mellinkoff, Sherman M. Idiopathic hypoparathyroidism with small bowel x-ray features of sprue, without steatorrhea. *Am. J. Digest. Dis.*, Mar., 1962, *7*, 273–280. (From: University of California Medical Center, Los Angeles 24, Calif.)

Idiopathic hypoparathyroidism is an uncommon disorder with a variety of well-described clinical features. Muscle cramps, tetany, seizures, papilledema, cataracts, ectodermal changes, and personality alterations are among the many variable presenting manifestations. Recently a curious association of a malabsorption state resembling sprue in patients who would otherwise appear to be suffering from either idiopathic or pseudohypoparathyroidism has been noted. The purpose of this article is to report a patient in whom this combination may have some bearing upon this problem.

Although low serum calcium levels may be caused by inadequate ingestion of vitamin D or calcium, acute pancreatitis, or chronic renal disease, the differential diagnosis of hypocalcemic states more frequently involves hypoparathyroidism and a malabsorptive disorder. The symptoms of diarrhea or steatorrhea in a hypocalcemic patient have favored the existence of a primary malabsorptive disorder. This has been considered evidence against the diagnosis of hypoparathyroidism.

It has become increasingly evident, however, that diarrhea, steatorrhea, and malabsorption of other substances occur with greater incidence in patients proved to have idiopathic or pseudohypoparathyroidism than would be expected on the basis of chance.

The severity of the symptoms related to the absorptive defect and the temporal relationship between the manifestations of malabsorption and those of hypoparathyroidism have been quite variable.

The findings in the present case would tend to support this view. The clinical features and response to parathormone were characteristic of idiopathic hypoparathyroidism. The initial hypoalbuminemia was probably related to the antecedent infection and was spontaneously corrected by the time the small-bowel roentgenograms were obtained. Yet there was a grossly abnormal appearance of the small bowel, resembling the changes seen in sprue, with marked improvement after correction of the hypocalcemia.

A decreased motility of the gastrointestinal tract as a cause of impaired absorption has been claimed by several workers.

An alternative explanation for the malabsorption in hypoparathyroidism is overgrowth of intestinal bacteria. The malabsorptive features of the "blind-loop" syndrome are seen secondary to a variety of intestinal lesions which dispose to pooling and stagnation of intestinal contents. It is reasonable to guess that this may have occurred in the sluggish and distended loops of bowel which appeared in the present case of hypoparathyroidism.—*Stephen N. Tager, M.D.*

Kaiser, E. Surgical aspects of non-inflammatory diseases of the stone-free gallbladder. *German Med. Monthly*, Jan., 1962, *7*, 18–22. (Address: I. Med. Klin., Med. Akademie, Düsseldorf, Germany.)

While the stone-free gallbladder rarely causes symptoms sufficiently severe to require cholecystectomy, in approximately 5 per cent in general, and in 2.2 per cent of the author's series of 1,500 cases, such stone-free gallbladders were removed surgically. In these cases there were many congenital anomalies of form and position of the gallbladder. Hormonal and metabolic disturbances in various combinations were also noted.

Usually, the pendulous gallbladder, associated with an intestinal ptosis is clinically of no significance. However, with a long mesentery it may become twisted and result in volvulus. If the fundus rests upon the colon it may simulate a "Phrygian cap." When adhesions are present this appearance may remain fixed, otherwise with contraction of the gallbladder this may disappear.

The speed of gallbladder evacuation depends upon the relationship between the force of the gallbladder musculature and the tone of the sphincter. The siphon-shape of the anatomic portions of the gallbladder, which occurs as a variant, may not obstruct evacuation but with additional anomalies, stasis, dilatation, inflammation, adhesions and stone formation may result. A recess (Hartmann's pouch) may become adherent to the duodenum and to the common duct.

While the "Phrygian cap" is a common variant noted before and after contraction, in some cases stasis and inflammation may occur in the sac behind the kind. Small folds and septa may vary considerably in number and depth. They occur more frequently in women than in men. These anomalies are not easily visualized at times. The clinical history is important but often difficult to evaluate.

Diverticula are congenital malformations of the gallbladder and are not acquired. They are pouches of the gallbladder wall with relatively small openings and are located at right angles to the fundus. They may be multiple. A narrow opening and weakness or even the occasional absence of muscle predisposes to stasis, inflammation and stone formation. Luschka's ducts or Rokitansky-Aschoff's sinuses are intramural diverticula or herniations of the mucosa through the muscularis. They may be present to a small extent in normal gallbladders but they may involve the entire gallbladder wall.

The strawberry gallbladder or cholesterosis is a metabolic disease of the stone-free gallbladder. It is believed to be due to increased cholesterol absorption of the mucosa. The clinical picture ranges from dyspeptic symptoms to a typical gallbladder colic. There is no characteristic cholecystographic appearance. Cholesterosis may lead to stasis and inflammation and eventually to cholelithiasis.—*J. N. Ané, M.D.*

Van Rossum, R. La biloptine: complément efficace de la cholécystographie de routine. (Biloptin: an efficient contrast medium complementary to routine cholecystography.) *J. belge de radiol.*, 1961, *44*, 389–419. (Address: Dr. R. Van Rossum, 53 Boulevard du Jubilé, Bruxelles, Belgium.)

Biloptine is the sodium or potassium salt of beta (3 dimethyleneamino—2, 4, 6 triiodophenyl) propionic acid. High solubility, rapid intestinal (duodenojejunal) resorption, low toxicity (excellent tolerance) and rapid excretion by the hepatic cells are important advantages. It is thus possible to visualize the biliary tract 2 hours after the ingestion and the gallbladder after the sixth hour and on. For a functional cholecysto-cholangiographic study, it is recommended to give 2 doses of 3.0 gm. each; one 10 hours and the other 3 to 4 hours prior to roentgenography (preferably in the morning for physiologic reasons). Cholecystectomized patients need take one dose of 6 capsules of 0.5 gm. each, only 1 or 2 hours before the examination.

Compared to other oral contrast media, biloptine produces a more homogeneous and clearer visualization of the gallbladder and a better evaluation of the physiology of the entero-porto-hepatic cycle is obtained. Compared to biligrafine (an intravenous cholangiographic medium), all oral media, in particular biloptine, are superior in demonstrating the biliary tract in patients with poor liver function (bromsulphalein retention of 30 per cent or more in 40 minutes) and with low serum protein. Biligrafine is dependent on the process of combination with serum protein in order to be concentrated in the liver and excreted subsequently. Oral media depend more on the entero-porto-hepatic cycle for concentration and biloptine is much more readily excreted by the hepatic cells. In 40 per cent of cases it is possible to demonstrate both the gallbladder and the common bile duct at the same time. Hence, the author of this detailed article suggests the use of biloptine in conjunction with other contrast media whether oral or intravenous.—*J. N. Sarian, M.D.*

Yeoh, K. S., and Ho, K. K. A case of carcinoma of the liver eroding into the extrahepatic bile duct demonstrated by transhepatic cholangiography. *Singapore M. J.*, Dec., 1961, *2*, 154–157. (From: Department of Surgery and Department of Pathology, University of Malaya, Singapore, Malaya.)

A case of carcinoma of the liver eroding into the wall of the common duct is described. Transhepatic cholangiography was used to demonstrate the filling

defect in the bile duct in this deeply jaundiced patient.

Carcinoma of the liver is known to cause jundice by infiltration of the organ and obstruction of the intrahepatic biliary ducts. It is, however, extremely uncommon to see carcinoma of the liver causing jaundice by obstructing the common bile duct.—*F. B. Markunas, M.D.*

Pierson, M. Antoine, and de Kersauson, M. C. Le diagnostic radiologique de quelques tumeurs abdominales chez l'enfant. (Radiologic diagnosis of some abdominal tumors in children.) *J. de radiol., d'électrol. et de méd. nucléaire*, Aug.–Sept., 1961, *42*, 433–446. (From: Clinique médicale infantile and Service central d'Électroradiologie du Centre Hospitalier Régional de Nancy, Nancy, France.)

This article is a review of roentgen diagnosis of abdominal tumors in children.

A few cases and fine roentgenograms are presented and practical considerations in diagnosis are discussed under four sections. These are: (1) urinary tract visualization; (2) retroperitoneal pneumography; (3) angiography; and (4) barium studies of the gastrointestinal tract. The chest roentgenographic findings are also discussed.

The importance of co-operation with the pediatrician (for diagnostic orientation) and methodic roentgen investigation is stressed.—*J. N. Sarian, M.D.*

Genitourinary System

Nordyke, Robert A. The radioisotope renogram: advances in test substance and procedure. *J. Nuclear Med.*, Jan., 1962, *3*, 67–75. (Address: Straub Clinic, Honolulu 14, Hawaii.)

Many previously used radioactive substances for obtaining renograms have not been entirely ideal. Removal by the liver and slow removal from the blood stream by the kidneys has created certain problems.

Hippuran (I^{131} labeled sodium o-iodohippurate) has eliminated some of these problems. This drug is removed from the blood only by the kidneys and is almost completely cleared from the blood with each passage through the kidneys. This shortens testing time. The qualities also allow for better collimation and the blood clearance rate approximates renal plasma flow.

Reproducibility of renograms and difficulties with distinguishing differences caused by renal pathology and by dissimilar geometry have created additional problems.

The author has employed (because of the characteristics of hippuran) collimators with a wider diameter probe (about $2\frac{1}{2}$ inches), a $2\frac{1}{2}$ by 6 inch rectangular skin surface, and a 1 inch crystal recessed 3 inches (later 6 inches) within the lead shield. This gives simplicity to placement of the collimators and therefore roentgenographic localization is not used. Three photographs of the system are shown.

The increased skin-to-crystal distance takes advantage of the fact that, at greater distance from the radioactive source, the geometric variation produces less difference in the count rate, and the wide skin field allows variable kidney positions (except intrapelvic), while still providing good shielding between the two kidneys.

A dose of 30 to 60 μc I^{131} labeled hippuran was used. Three figures illustrating the results in normals are shown. These results reveal no difference from those obtained with the usual collimators having a $1\frac{1}{2}$ inch aperture, $1\frac{1}{2}$ inch recession of the crystal, and roentgenographic localization. Exclusion of radioactivity from the opposite kidney is said to be excellent with the author's collimator system. Improved reproducibility and comparability of curves in the presence of equally functioning kidneys were obtained.

The author concludes that it is still hazardous to estimate total renal function by the renogram except where gross abnormality is present. However, improved materials and instrumentation have brought the renogram to a state of high sensitivity and reliability when used to compare one kidney with the other.—*William K. Littman, M.D.*

Schwartz, Franklin D., and Madeloff, Michael S. The use of radiohippuran in the diagnosis of unilateral renal disease. *J. Urol.*, Mar., 1962, *87*, 249–257. (From: The Division of Renal Disease and Hypertension of the Department of Medicine, University of Maryland School of Medicine, and the University Hospital, Baltimore, Md.)

Simultaneous renal clearances of radiohippuran and para-aminohippuric acid (PAH) were performed in 25 hypertensive patients representing a variety of unilateral and bilateral disease processes, along with others showing normal renal function. The average ratio of radiohippuran clearance to PAH clearance was 0.81 with a wide range of 0.5 to 1.2. This ratio, however, was quite constant for each patient over a number of collection periods, and the differences in clearances between the two sides in an individual patient were predominantly identical for the two substances. The lack of agreement of the two clearances with the low doses used may be due to competitive tubular inhibition or to the excessive protein binding of radiohippuran.

Radioactive renograms were obtained within 72 hours after performance of differential clearances in 6 patients with unilateral renal disease and in 1 with

normal renal function. In each instance where a depressed vascular phase was present unilaterally, there was an associated decrease clearance of PAH and radiohippuran on that side. There was excellent agreement between the alterations in differential clearance findings and the differences in the magnitude of the vascular phase of the renogram.

In 2 patients with a solitary kidney simultaneous renography and plasma clearances of radiohippuran showed that the greatest rate of plasma clearance coincided with the peak of the vascular phase.

The authors have shown that in patients with unilateral arterial disease of the kidney or unilateral pyelonephritis there is a significant lowering of PAH clearances. The renogram coincides with the measurement of radiohippuran clearances, and therefore is an ideal screening test for detecting curable hypertensive disease of unilateral renal origin. The possibilities of false interpretation of the radiohippuran renogram are discussed.

The authors include 5 tables and 8 figures which graphically illustrate the text of the article.—*George W. Chamberlin, M.D.*

CAMERON, J. R. Bilateral hereditary polycystic disease of the kidneys associated with bilateral teratodactyly of the feet: "pied en pince de homard." *Brit. J. Urol.*, Dec., 1961, *33*, 273–475. (Presented at the Seventeenth Annual Meeting of the British Association of Urological Surgeons at Edinburgh, June, 1961.)

In a significant number of patients with polycystic disease of the kidneys, there are congenital and hereditary anomalies elsewhere in the body. The author reports a case of bilateral polycystic disease of the kidney and a pronounced form of teratodactyly with bilateral cleft feet.

Both conditions are considered to be inherited in dominant manner and, in the case presented, the mother, a brother, and two nieces have extremity deformities. Complete family investigation could not be carried out. The brother with foot involvement and hypertension, a blood urea nitrogen of 31 mg. per cent and albuminuria, has since then died and there is no other knowledge of his renal status.—*Lois Cowan Collins, M.D.*

JORDAN, WILLIS P., JR. Peripelvic cysts of the kidney. *J. Urol.*, Feb., 1962, *87*, 97–101. (From: The Department of Urology, Veterans Administration Hospital, Lake City, Fla.)

Peripelvic cysts are defined as simple or multiloculated cysts situated in the hilus of the kidney and not connected with the excretory passages but producing various deformities of the calyceal system or renal pelvis. Their etiology is unknown but some writers have considered them to be of congenital origin, whereas others believe that they may be associated with inflammatory obstruction. These cysts are relatively uncommon, but of all cases of cystic disease of the kidney they may represent an incidence of approximately 6 per cent.

The author presents 4 detailed case reports with roentgenographic studies including aortography, nephrography, and pyelography to show the various defects which may occur in the calyceal system or renal pelvis as a result of peripelvic cysts. The defects are usually of a smooth concentric type, distorting the pelvis medially or projecting into the lumen of the infundibulum of the calyx. The differential diagnosis includes carcinoma of the renal parenchyma, renal pelvic carcinoma, and cyst of the renal parenchyma. Exploration of the hilar mass is recommended to substantiate the diagnosis and to remove the pressure effects of the cyst. Six composite illustrations are included in this article.—*George W. Chamberlin, M.D.*

HEYMAN, ARNOLD, and LOMBARDO, LOUIS J., JR. Metastatic prostatic abscess with report of a case in a newborn infant. *J. Urol.*, Feb., 1962, *87*, 174–177. (From: The Los Angeles County Hospital, Los Angeles, Calif.)

This is a case report of a 34 day old infant with a large metastatic abscess of the prostate which produced obstruction of the urethra. On admission to the hospital, the history indicated that the mother had a breast infection while nursing. The patient's temperature was 102.6° F., pulse 124, respirations 30, hemoglobin 12.3 gm., and the white blood cell count 37,000. The urinalysis showed an occasional red and white blood cell. A growth of *Staphylococcus albus* was obtained and roentgenograms of the chest revealed signs of a right upper lobe pneumonia which was treated. During the hospital stay, there were episodes of diarrhea and the blood culture showed *Staphylococcus albus*, coagulase negative. The patient responded to treatment and was discharged from the hospital.

Three days later, he returned because of swelling and tenderness in the left scrotum. A firm, tender, globular mass, three to four times the size of the normal scrotal contents, occupied the left scrotum. It did not transmit light. The epididymis could not be distinguished from the testis, and the entire mass was warm to palpation. The white blood cell count was 24,000 and the blood urea nitrogen was 13 mg. per cent. A working diagnosis of funiculitis and epididymo-orchitis was made. An excretory urogram revealed mild right hydronephrosis and hydroureter. Following this, the abdomen became distended and the survey roentgenogram showed most of the opaque medium retained in the markedly distended bladder. Two hundred cc. of urine was obtained on catheterization, and shortly afterward another 220 cc. of

urine was withdrawn. The urine culture showed *Staphylococcus albus*, coagulase negative.

Rectal examination revealed an enlarged, fluctuant prostate. During an attempted cystoscopic examination, the instrument punctured the prostate and a large volume of purulent drainage occurred. Culture of this drainage showed *Staphylococcus aureus*, coagulase positive. The patient was then placed on catheter drainage and treated with sulfanilamides and antibiotics, blood transfusions, cortisone, and vitamins and iron. His clinical condition improved and a second excretory urogram showed less evidence of obstruction. The patient left the hospital afebrile and gaining weight.

He subsequently returned about 6 months later complaining of diarrhea. Rectal examination at this time was normal and the roentgenograms of the chest showed right upper lobe pneumonitis which responded to therapy. A urethrogram disclosed no abnormality and the excretory urogram revealed normal findings in the upper urinary tract.

The authors discuss the etiology of metastatic prostatic abscess in infants and emphasize the need for routine rectal examination in all pediatric patients. Two composite illustrations show the roentgenographic changes which occurred during the disease process.—*George W. Chamberlin, M.D.*

Clifford, Nathan J., and Katz, Isadore. Subcutaneous emphysema complicating renal infection by gas-forming coliform bacteria: a report of two cases in diabetic patients. *New England J. Med.*, Mar., 1962, *266*, 437–439. (From: The Departments of Medicine and Radiology, State University of New York, Downstate Medical Center, and the Kings County Hospital Center, New York, N.Y.)

In a case reported of a 70 year old man, presumably the urinary-tract infection was introduced by catheterization after the herniorrhaphy. The overwhelming gas-producing renal infection spread to the perinephric space, dissected through adjacent muscle, and entered the subcutaneous tissues of the abdomen and chest. Gas was also present in the urinary bladder. Pneumaturia was observed on one occasion.

It is generally agreed that the formation of gas in the tissues and body fluids of diabetic patients is due to the fermentation of glucose by organisms that produce carbon dioxide from glucose *in vitro*. The organisms isolated from the patients reported were avid gas formers *in vitro*. In diabetes, infection with these organisms has produced retroperitoneal abscess, perirectal abscess, gaseous infiltration of the renal and perirenal tissues, gas-containing vesicles in the wall of the urinary bladder, pneumaturia, and subcutaneous emphysema. The 2 patients presented are unique in that a gas-forming renal infection spread to the *subcutaneous tissues*.

The subcutaneous emphysema noted differed from that seen in gas gangrene and also from the subcutaneous emphysema of respiratory origin. The skin was slightly edematous but not discolored or necrotic as it often is in gas gangrene.

The occurrence of a gas-forming coliform infection in diabetic patients is an ominous sign. Nearly all the reported cases with extensive involvement have terminated fatally. Extensive incision of the affected tissues has been the only effective treatment in patients with subcutaneous emphysema of infectious origin. Efforts to control the diabetes before surgical intervention are not effective since the necrotizing infection renders adequate control impossible and will spread rapidly unless prompt drainage is instituted.

In both the reported cases, the infection occurred in patients with diabetes mellitus.—*Stephen N. Tager, M.D.*

Leadbetter, Guy W., Jr., and Leadbetter, Wyland F. Urethral strictures in male children. *J. Urol.*, Mar., 1962, *87*, 409–415. (From: The Massachusetts General Hospital and Harvard Medical School, Boston, Mass.)

Strictures of the male urethra in children are relatively uncommon. They may be congenital, infectious or traumatic in origin. The symptoms are those of obstruction and in the congenital type often do not become obvious until the age of 2 or 3. The diagnosis is best confirmed by cysto-urethrography. It is important that the roentgen ray evaluation be done prior to any sounding or dilatation, so that the exact degree and extent of the lesion can be demonstrated. Characteristic findings on the retrograde urethrogram show dilatation distal to the area of stenosis, while on a voiding cystourethrogram the dilatation is proximal to the stenotic region. Direct visualization of the stricture with the infant panendoscope may allow an accurate estimation of the degree and extent of the stricture prior to dilatation. The complications of untreated or inadequately treated urethral stricture may be chordee, chronic prostatitis, epididymitis, contracture of the bladder neck, decompensated bladder with residual urine, pyelonephritis, vesical calculus, hydronephrosis, renal damage and even death.

This article includes case histories of 6 children with ureteral strictures and 2 adults in whom the strictures developed following an accident in their youth. All of these patients had adequate trial of dilatation and have subsequently been treated by formal surgical procedures. Five patients had a two stage Johanson urethroplasty, 2 have had the first stage of a similar urethroplasty, and 1 had a single stage procedure.

The surgical technique is detailed and the case histories are given in abstract. Six composite illustrations show the various lesions of the urethra before

and after surgical correction. The results of this procedure have been satisfactory.—*George W. Chamberlin, M.D.*

Calatroni, Carlos J., Poliak, Aaron, and Kohan, Angel. A roentgenologic study of stress incontinence in women. *Am. J. Obst. & Gynec.*, Mar., 1962, *83*, 649–656. (Address: Helguera 4284, Buenos Aires, Argentina.)

The problem of stress incontinence was studied in 115 women. Using a probe, a flexible chain of opaque beads was inserted into the urethra. One end of the chain was left in the bladder, the other end was taped to the thigh. The bladder was then filled with 60 cc. of 10 per cent sodium iodide. With the patient in the erect position serialographic roentgenograms were taken in frontal and lateral projections while the patient was straining.

Three types of urethrovesical prolapses were found. In the first type, a cystocele was present but the internal meatus of the urethra was situated above and anterior to the lowest part of the cystocele. Stress incontinence was very rare in this group because the greatest urostatic pressure acts behind the meatus.

In the second type of urethrovesical prolapse, the floor of the bladder had a funnel shape and it was impossible to determine where the urethra begins and the bladder ends. There is a loss of the posterior urethrovesical angle but the authors feel that this is of secondary importance to the formation of the urethrovesical funnel. The distance between the urethra and pubis is greater than in type one since the proximal urethra is displaced downward and posteriorly. In this group the greatest urostatic pressure acts upon a deficient sphincter and there is stress incontinence. No cystocele is present. This type accounted for 95 per cent of the patients with incontinence.

The third type of urethrovesical prolapse was a combination of types one and two, the patient having both a cystocele and a funnel-shaped urethrovesical junction. These patients are potentially incontinent and loss of urine will occur if the cystocele is repaired without correction of the abnormal urethrovesical junction. In order to prevent incontinence the internal meatus of the urethra should be elevated and brought forward at the time of surgery so as to displace it from the point of maximum urostatic pressure.—*J. L. Williams, M.D.*

Green, Thomas H., Jr. Development of a plan for the diagnosis and treatment of urinary stress incontinence. *Am. J. Obst. & Gynec.*, Mar., 1962, *83*, 632–648. (Address: 226 Marborough Street, Boston, Mass.)

The author's experience with 152 patients suffering from stress incontinence is summarized and a plan for evaluation and treatment is presented. Adequate investigation is necessary to differentiate true stress incontinence from other conditions which may closely simulate it. The most important study is a urethrocystogram using a string of metallic beads in the urethra and an opaque medium in the bladder.

Loss of the posterior urethrovesical angle is a constant feature in cases of stress incontinence, and restoration of the normal angle is essential if the patient is to be cured by surgery.

Study of the urethrocystograms also demonstrates that in some patients the axis of the urethra is greater than 45° from the vertical. This is due to posterior and downward displacement of the proximal portion of the urethra and results from injury to the supports of the urethra and the bladder neck. Such patients invariably present the most severe degree of stress incontinence and are the most difficult to treat. Vaginal repair alone will not restore the normal urethral axis and incontinence will persist in more than half of the cases unless a suprapubic urethrovesical suspension is also done.

Careful attention to the preoperative urethrocystograms, with especial attention to the axis of the urethra, has permitted a better choice of the optimum operative procedure and has increased the cure rate to 93 per cent.—*J. L. Williams, M.D.*

Dardari, M., Santino, A., Brunelli, B., and Amici, F. Metodologia, indicazioni e resultati clinici dello studio radiologico dei surreni ("pneumosurrenografia"): rassegna sintetica e contributo casistico. (Methods, indications and clinical results of radiologic study of the adrenals ["pneumoadrenography"]: synthetic analysis and casuistic contribution.) *Ann. radiol. diag.*, 1961, *34*, 298–342. (From: Istituto di Radiologia dell' Università, Modena, Italy.)

The purpose of this report is to emphasize the advantages and limitations of "pneumoadrenography" in pathologic conditions of the adrenal glands. Cases that are clinically suspected of having disturbed function of the adrenal glands are first studied by conventional roentgenographic methods: the skull for changes of the sella turcica; the chest for primary malignancy or metastatic disease; the gastrointestinal tract for displacement or deformity caused by retroperitoneal tumors; and intravenous urography for primary kidney tumors or displacement and encroachment from tumors of adjacent organs.

The procedure for "pneumoadrenography" is described. After injection of 1,500–1,800 cc. of air laminagrams are taken in anteroposterior projection from 5 to 8 cm. above the surface of the back. In addition, lateral laminagrams 4 to 6 cm. from midline for the right and left adrenal glands are taken. Thus the adrenal glands can be seen in two dimensions to determine their size and shape. For interpretation,

one must have a thorough knowledge of the normal appearance, and the normal variation of the uninvolved adrenal glands. The diseased adrenal gland may change in size, shape, or density. Sometimes calcium deposits are present. An enlargement of the gland, or increase in density or distortion of its triangular shape may be detected by "pneumoadrenography."

Cases, with history and illustrations, are presented of adrenogenital syndrome; Cushing's syndrome; paroxysmal hypertension; and Addison's disease.

In adrenogenital syndrome, if unilateral or bilateral enlargement of the adrenal glands can be demonstrated, medical treatment is indicated. If the study shows presence of a tumor then surgery is performed.

In Cushing's syndrome due to an abnormal adrenal gland, these studies will be useful to determine to which side the surgical intervention should be directed.

In paroxysmal hypertension the desired information is whether the tumor is unilateral or bilateral, single or multiple or whether it is located in an aberrant region. The same holds true for malignant tumors of the adrenal gland.

In Addison's disease this information is not necessary, since the clinical and laboratory data confirm dysfunction of the adrenal glands.

The limitations of this procedure are that disturbed function may be present without morphologic changes of the adrenal glands and that it is not always possible to localize an aberrant pheochromocytoma. In cases where the air fails to envelop the adrenal gland, malignant involvement should be seriously suspected. Tumors of adjacent organs at times may not be differentiated from adrenal tumors.

The authors conclude that "pneumoadrenography" has a useful place in the study of adrenal gland malfunction and tumors.—*Peter E. Russo, M.D.*

Skeletal System

Donaldson, William F., Jr. (Pittsburgh, Penna.) Aneurysmal bone cyst. *J. Bone & Joint Surg.*, Jan., 1962, *44A*, 25–40.

In this article the author reviews the literature on aneurysmal bone cyst. Six new cases are evaluated which were previously diagnosed as bone cysts, giant-cell variant, and atypical giant-cell tumors since 1950 at the Children's Hospital and Presbyterian Hospital in Pittsburgh, but have been recently reclassified as aneurysmal bone cysts. Aneurysmal bone cyst is a benign lesion occurring primarily under the age of 30 with no preference for sex. It has been reported in almost every bone of the skeleton, but chiefly in the spine and long bones.

The gross lesion is expansile in character being delineated by subperiosteal new bone and filled usually with a pulpy red tissue that bleeds freely. At times the lesion may be primarily a fluid-filled cyst with only scanty tissue. Microscopically, the tissue in these lesions appears the same. Septa are found about vascular spaces. These septa are composed of active fibrous tissue, various numbers of foreign body giant cells and osteoid. The vascular spaces vary greatly in size, have no muscular coat and often do not have an endothelial lining. These spaces contain normal-appearing erythrocytes.

The author believes that his material confirms that these cysts represent some variation in vascular supply or hemodynamics rather than a secondary manifestation developing in a pre-existing lesion altered by a superimposed pathologic process.

Roentgenologically, aneurysmal bone cysts pass through several stages. The lesion is confined to the cortex and medullary portion of the bone and appears to erode these areas first. Later the periosteum is raised giving an expansile "bubble-like" appearance. In the late stages the lesion may lose its shell of subperiosteal bone.

In the long bones, one first finds a lytic lesion, usually eccentric in the metaphyseal area which develops an expansile extracortical mass covered by subperiosteal new bone. A raised triangle of new bone may be found (Codman's triangle).

The posterior elements are characteristically involved in the spine with an expanded spinous process and laminae. There is the typical thin shell of subperiosteal bone outlining the mass.

In flat bones, these lesions show an expansile ovoid involvement that is quite symmetrically outlined by subperiosteal new bone.

The author feels that the treatment of choice is currettage. Roentgen therapy has been successful with reported doses of 600–2,000 r.—*George A. Miller, M.D.*

Sethi, Rajindra Singh, Climie, Andrew R. W., and Tuttle, William M. Fibrous dysplasia of the rib with sarcomatous change. *J. Bone & Joint Surg.*, Jan., 1962, *44A*, 183–188. (From: The Departments of Pathology and Thoracic Surgery, Harper Hospital, Detroit, Mich.)

A review of the literature by the authors revealed only 5 well-documented cases of sarcoma developing in fibrous dysplastic lesions, all but one being of the polyostotic variety.

An additional case, that of a 52 year old woman, is reported where a sarcomatous change developed in a solitary fibrous dysplastic lesion in a rib. The symptoms were primarily local pain, shortness of breath, anorexia and weight loss of one month's duration. Roentgenograms showed an intrathoracic, extrapleural soft tissue mass with destruction of the superior aspect of the anterior end of the left fifth rib and expansion of the shaft. The original biopsy was diagnosed as fibrous dysplasia. The mass was subsequently removed along with the involved rib. The

pathologic examination revealed an area of fibrous dysplasia in the rib, a transitional zone (showing more osteoid production and calcification of osteoid with larger and rather hyperchromic nuclei of the fibroblasts), and a large mass characteristic of osteogenic sarcoma.

The patient succumbed of her disease about 6 months after surgery and postoperative irradiation. —*George A. Miller, M.D.*

Goldenberg, Raphael R. Neoplasia in Paget's disease of bone. *Bull. Hosp. Joint Dis.*, Apr., 1961, *22*, 1–38. (From: The Orthopaedic Department, St. Joseph Hospital, Paterson, N. J.)

The incidence of sarcomatous degeneration in Paget's disease is difficult to assess because most reported cases have occurred where there has been advanced skeletal involvement, and uncomplicated Paget's disease is infrequently reported. In cases of extensive involvement the sarcomatous change is approximately 10 per cent, according to Lake. Considering all the cases of Paget's disease, the overall incidence can be estimated at less than 2 per cent.

The author believes that when a skeletal malignant degeneration develops in a person of 50 years or more, a complication of unrecognized Paget's disease should be suspected. In review of the literature about 80 per cent of the cases were in males. Uncomplicated Paget's disease shows little if any predilection of sex.

The reason for tumor formation occurring with Paget's disease is not known but a definite causal relationship appears to exist. Pain and deformity may be present with uncomplicated Paget's disease, but continual, increasing pain associated with a mass may denote developing sarcoma.

Biochemically, the serum calcium and phosphorus levels are nearly always normal but the alkaline phosphatase level is elevated. A stationary level implies static disease whereas a steadily rising level denotes progression of the disease. If a sarcomatous change supervenes and there are osteogenic potentialities, the level of serum alkaline phosphatase will usually be elevated. Newton reported the magnesium content of Paget bone to be higher than normal.

Roentgenograms may show changes of Paget's disease in the absence of pertinent clinical findings. Both rarefactions and sclerosis may be present. Malignant degeneration may produce areas of radiolucency in a previously altered bone, may break through the cortex, and may contain ossification within osteolytic areas. However, positive diagnosis of malignancy must be substantiated by biopsy.

The duration of the pagetic changes in bone or the extent of involvement is not a primary factor in the sarcomatous degeneration. The type of the malignant change can be a fibrosarcoma, osteogenic or chondrosarcoma, or a benign or malignant giant cell tumor.

Trauma appears to point to an already existing malignancy rather than to precipitate it. The prognosis of a sarcoma complicating Paget's disease is poorer than that usually ascribed to osteogenic sarcoma. Treatment is symptomatic and palliative.

The histories of 9 cases are presented. The age range was from 46 to 72 years. The lesions developing in areas of Paget's disease varied from osteogenic sarcoma to chondrosarcoma and in 1 case there was evidence of all three types of sarcoma and of giant cell tumor.—*George A. Kling, M.D.*

Karlen, Anders. On cystic tuberculosis of bone. *Acta orthop. scandinav.*, 1961, *31*, 163–177. (From: The Department of Orthopaedic Surgery, University of Malaya, Singapore, Malaya.)

The author reports 6 cases of cystic tuberculosis of bone occurring in young children from $2\frac{1}{2}$ to 4 years of age. Three of the cases consisted of solitary well defined metaphyseal or metaphyseal-epiphyseal cysts that clinically could not be differentiated from other cystic lesions. The remaining 3 cases represented the type of tuberculous skeletal manifestation called "multiple pseudocystic tuberculosis of bone".

The solitary cysts were preoperatively diagnosed as Brodie's abscess. This diagnosis was based upon the history and the roentgenologic appearance which showed a cystic lesion well bordered with a sclerotic zone, and in 1 case contained a sequestrum. In 2 of the cases the lesion occurred in the femoral neck and in 1 in the lower tibial metaphysis. The length of the history varied from 5 to 12 months. Clinically, pain, limp, and swelling were noted. The chest roentgenograms showed evidences of the primary complex. All 3 cases had curettage and the biopsy reports were positive for tuberculosis. They were treated by fixation and with streptomycin, PAS and INH with good results.

In the author's series of multiple pseudocystic tuberculosis of bone, the spine, mastoid, malar bone, scapula, ribs and skin were involved in addition to the long and short bones. At curettage the biopsy reports were positive for tuberculosis. One patient died following mastoidectomy. The remaining 2 cases were treated by fixation and by the administration of streptomycin, PAS and INH with good results. Some kyphosis in the thoracic area remained.—*J. N. Ané, M.D.*



Figure 1. *Preoperative radiograph:* Shows bilateral dislocation.

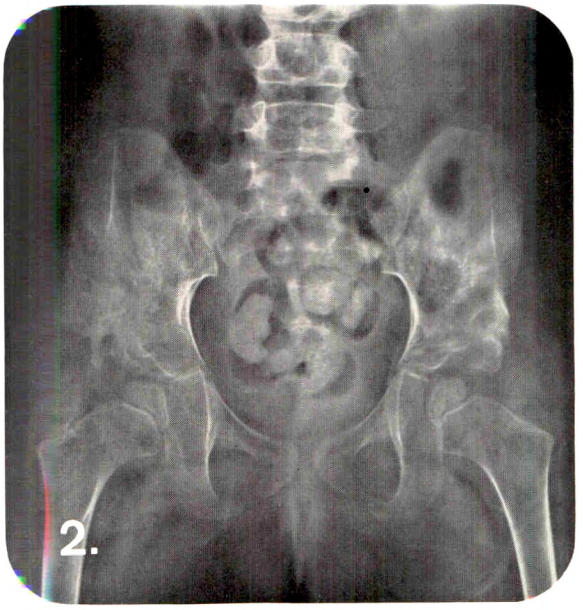


Figure 2. *Postoperative radiograph:* Hips in reduced position with well-constructed acetabula.

OPEN REDUCTION AND PEMBERTON OSTEOTOMY IN A CHILD 4 YEARS OF AGE with a previously untreated congenital dislocation of both hips

Preoperative and postoperative radiographs on Kodak Royal Blue medical x-ray film. Surgery photographed on Kodak Ektachrome roll film.

The case that follows is an excellent example of careful medical documentation. Note how well the essential surgical steps are covered; how effectively color delineates tissue differentiation, establishes the continuity of surgical action.

Above all, note how clearly radiographs and photographs combine to highlight the procedure—make it completely meaningful, from start to final radiograph, for all who see and read or hear.

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Note: Due to space limitations, outline of surgical procedure has been confined to left hip. Surgery of right hip duplicated that of left.

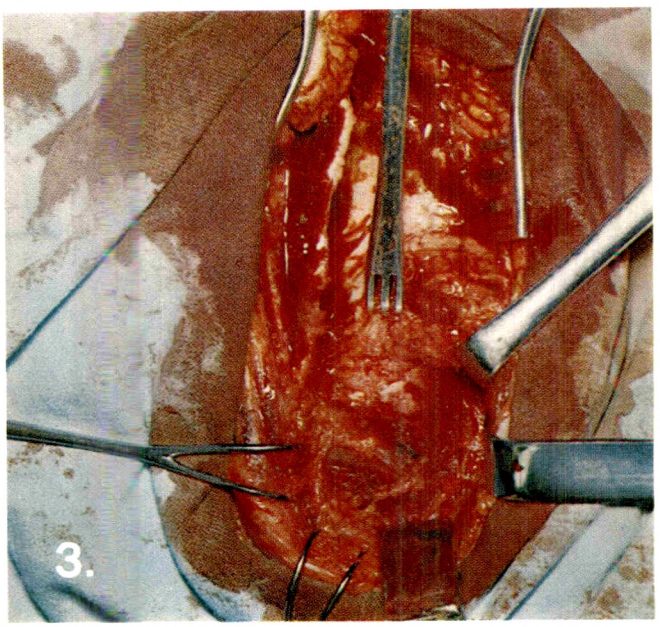
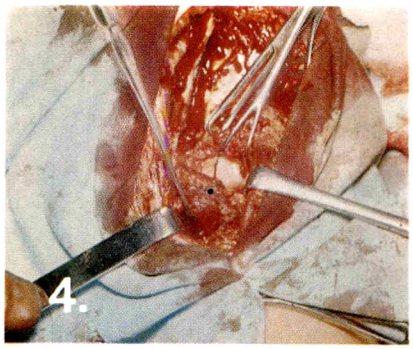
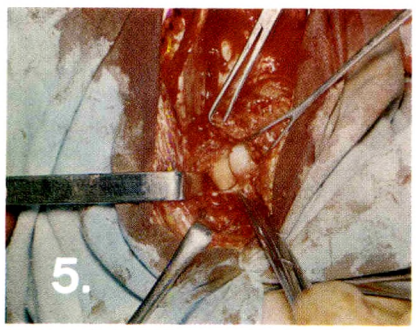


Figure 3. Exposure of left hip. Stripped anterior wing of the ilium above. Rectus being retracted laterally; iliacus fibers medially; capsule in between.

Turn page for balance of surgical procedure.





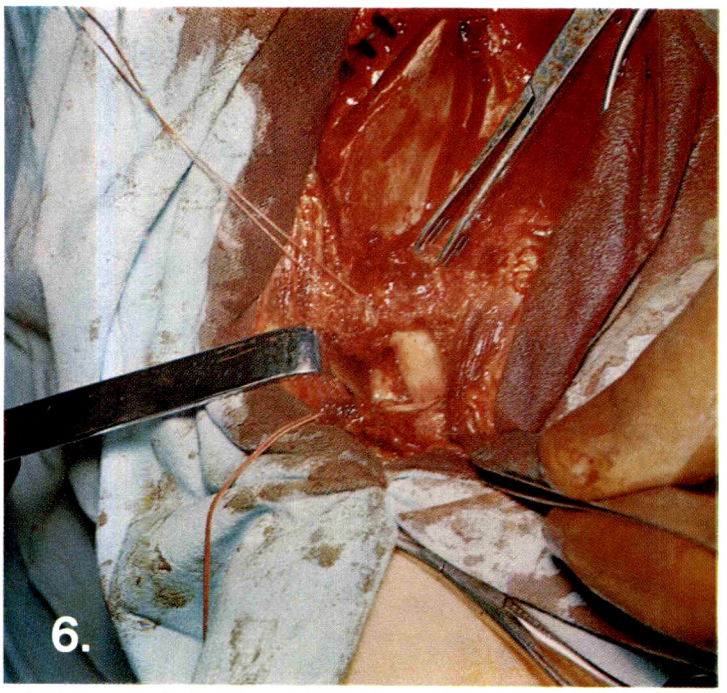
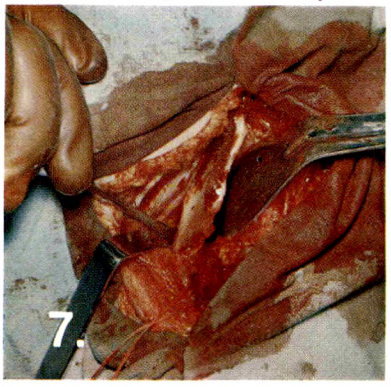
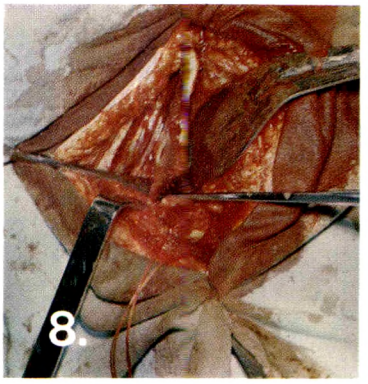


Figure 4. Capsule has been opened. Iliopsoas partly obscures acetabular cavity. Femoral head appears normal. **Figure 5.** Iliopsoas retracted medially to expose entrance to acetabulum. Roll of tissue obstructing entrance made up of deformed and thickened limbus. **Figure 6.** Iliopsoas has been cut in Z-fashion. Ends are separated and held by sutures for better visualization of the acetabulum. Thickened limbus clearly shown.





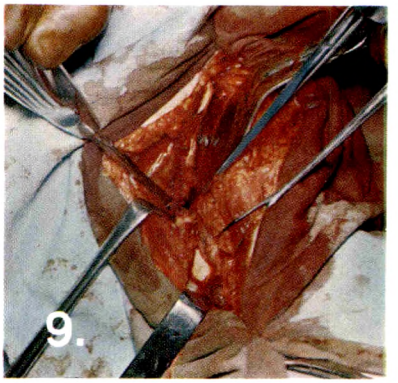
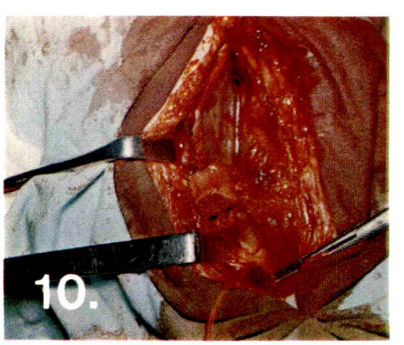


Figure 7. Inner table of ilium has been stripped by detaching iliac apophysis and retracting it with abdominal muscles and iliacus origin medially. **Figure 8.** An osteotome is opening an osteotomy which follows contour of roof of acetabulum to triradiate cartilage (Pemberton procedure). **Figure 9.** Hip has been reduced and osteotomy is being opened to make hip stable. **Figure 10.** Wedge of bone (from bone bank) has been driven into osteotomy to hold it open. Hip has been dislocated to show effect of osteotomy on acetabulum.

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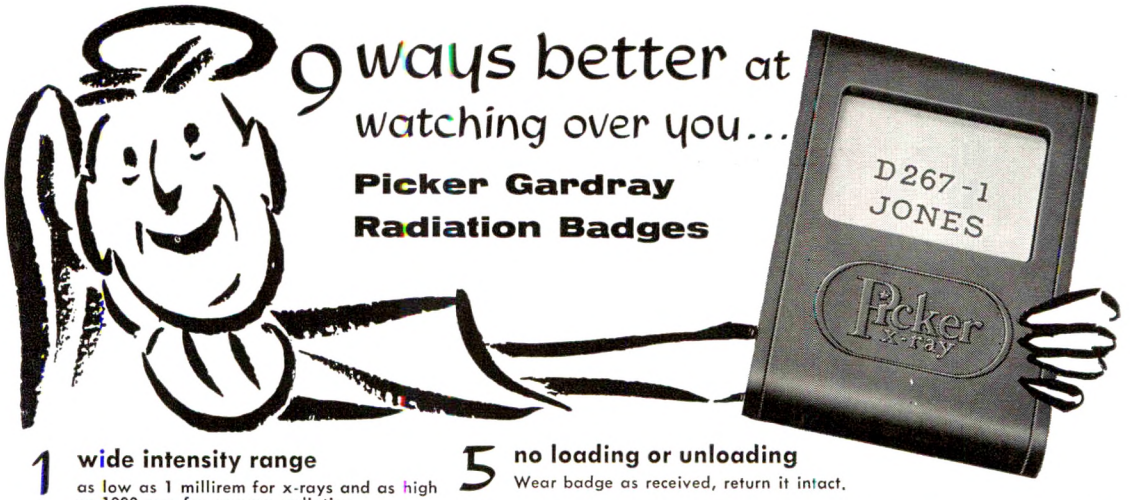
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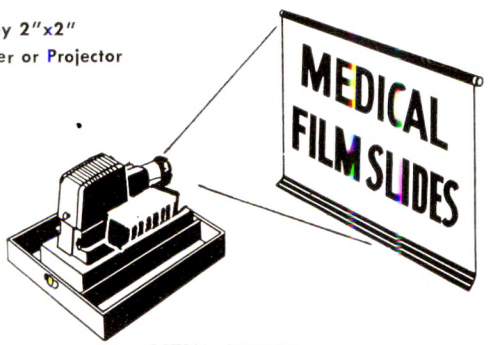
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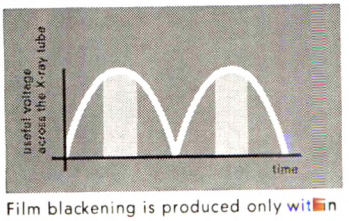
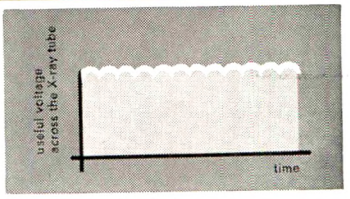
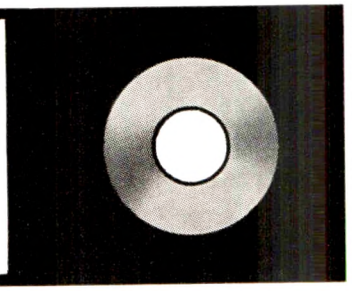
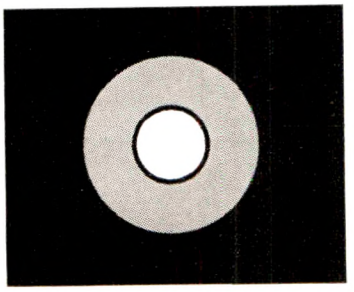
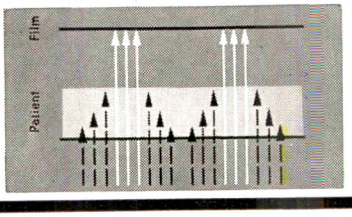
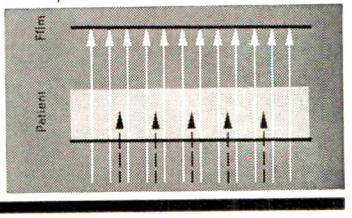
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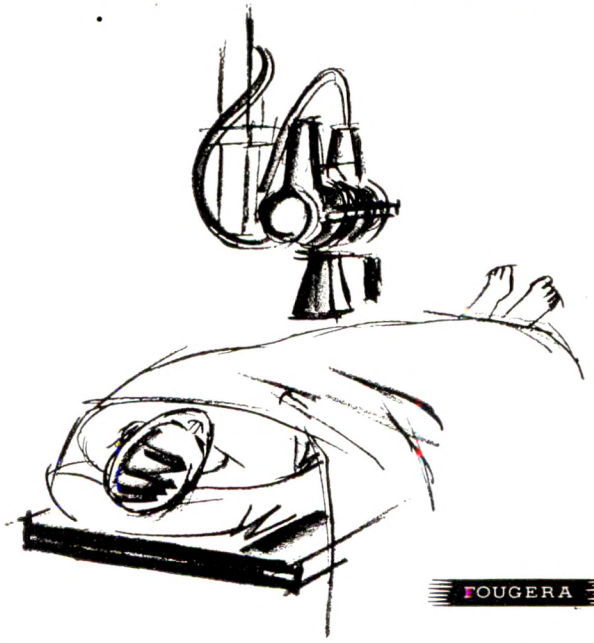
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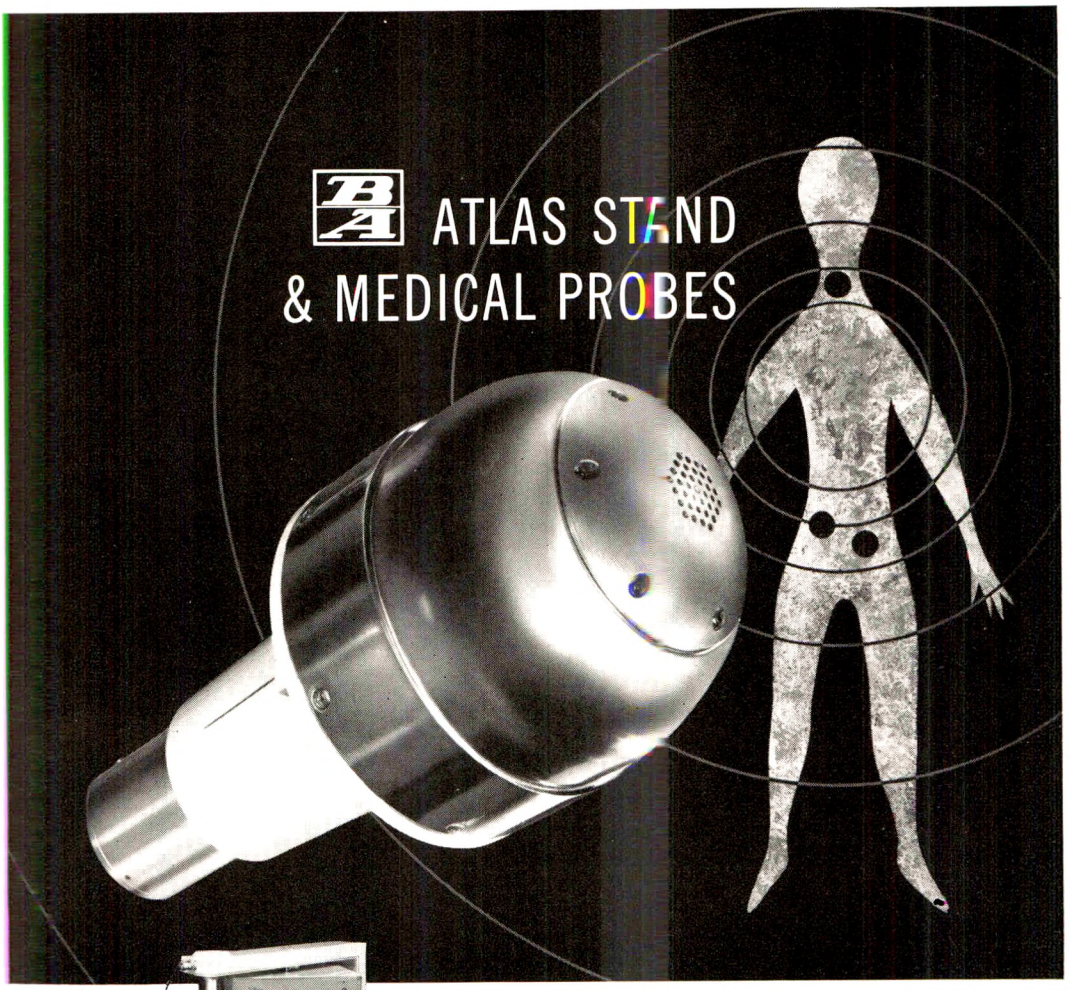
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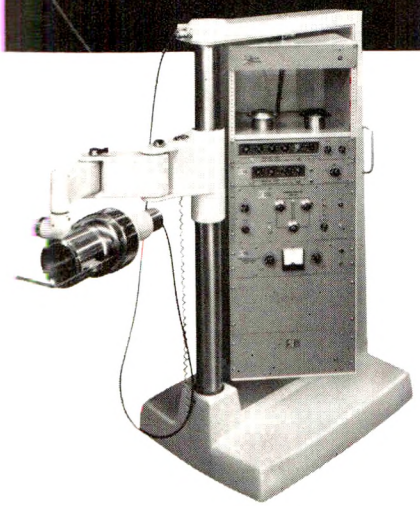
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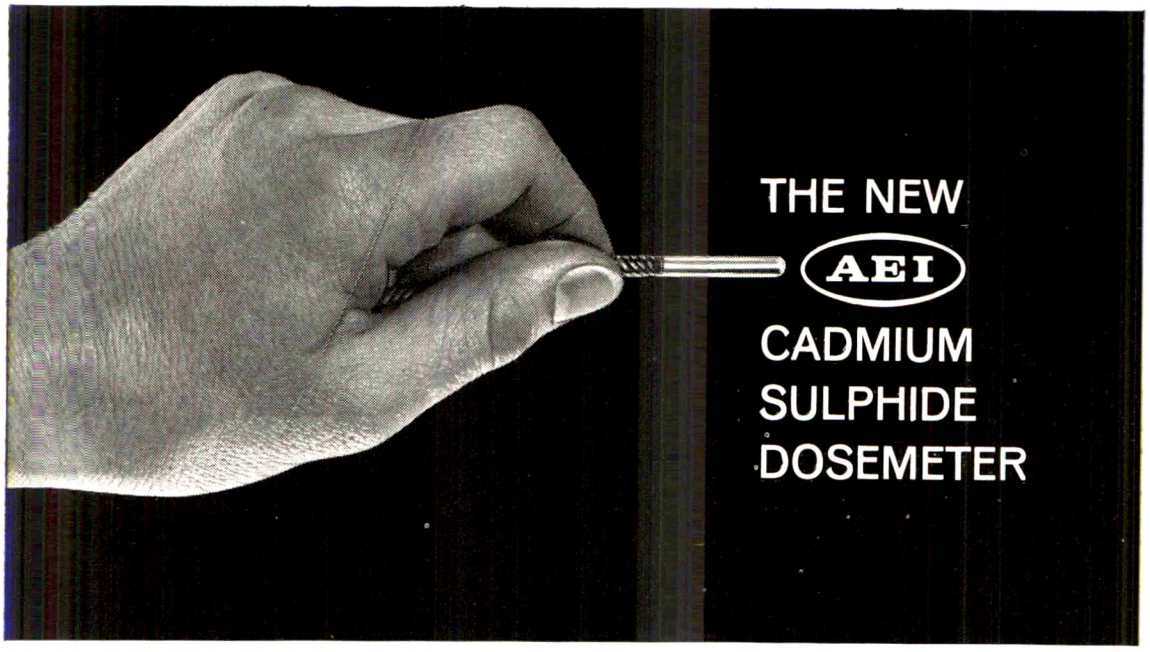
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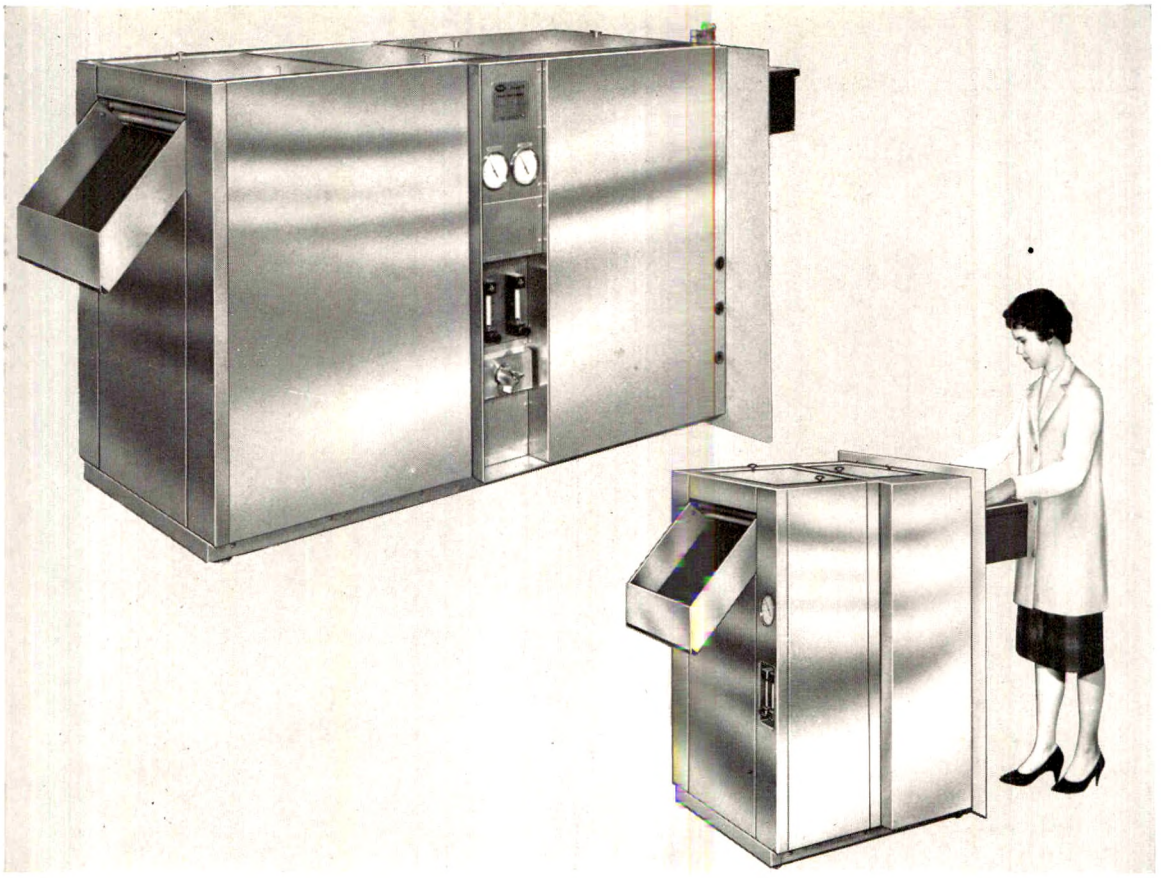
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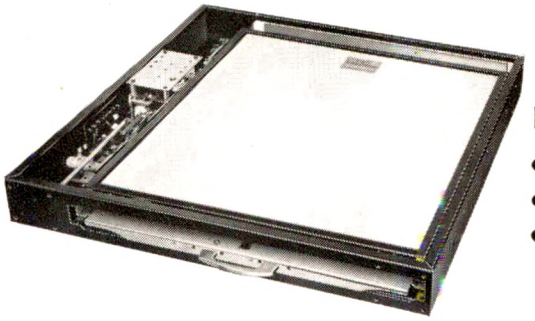
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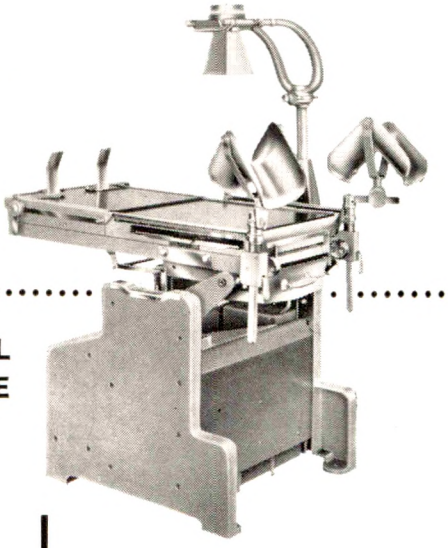


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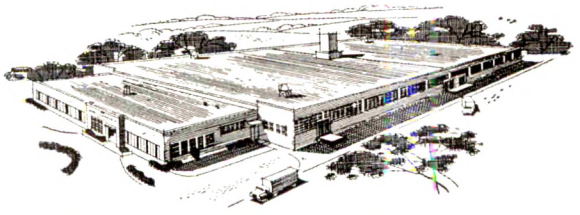
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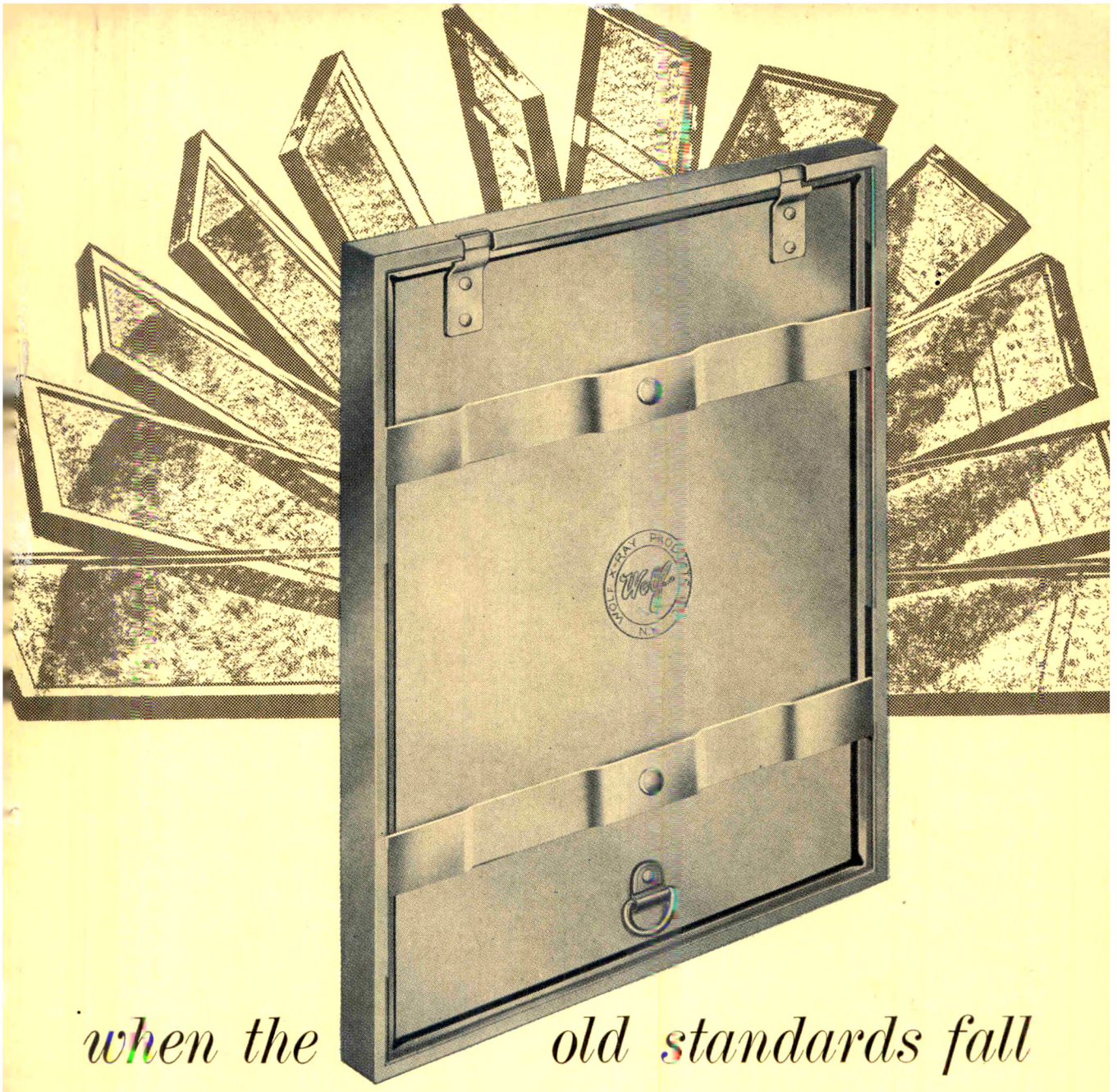
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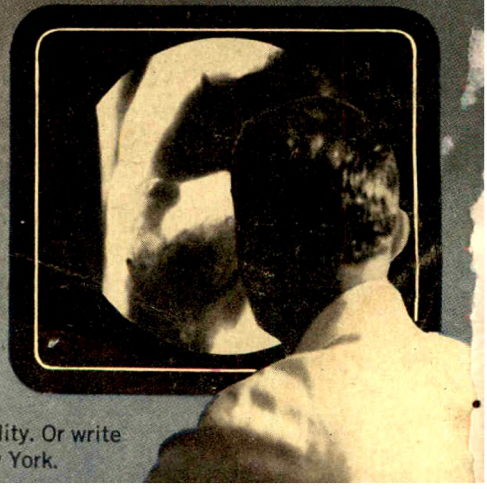


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